

**PROCEEDINGS**  
OF THE  
**ROYAL SOCIETY OF MEDICINE**

EDITED BY  
SIR JOHN Y. W. MACALISTER  
UNDER THE DIRECTION OF  
THE EDITORIAL COMMITTEE

**VOLUME THE SIXTEENTH**  
SESSION 1922-23

**PARTS I & II**

GENERAL REPORTS

SECTIONS:—

ANÆSTHETICS	BALNEOLOGY AND CLIMATOLOGY
STUDY OF DISEASE IN CHILDREN	CLINICAL DERMATOLOGY
ELECTRO-THERAPEUTICS	EPIDEMIOLOGY AND STATE MEDICINE
HISTORY	MEDICINE
LARYNGOLOGY	NEUROLOGY



LONDON  
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1923



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GENERAL REPORTS



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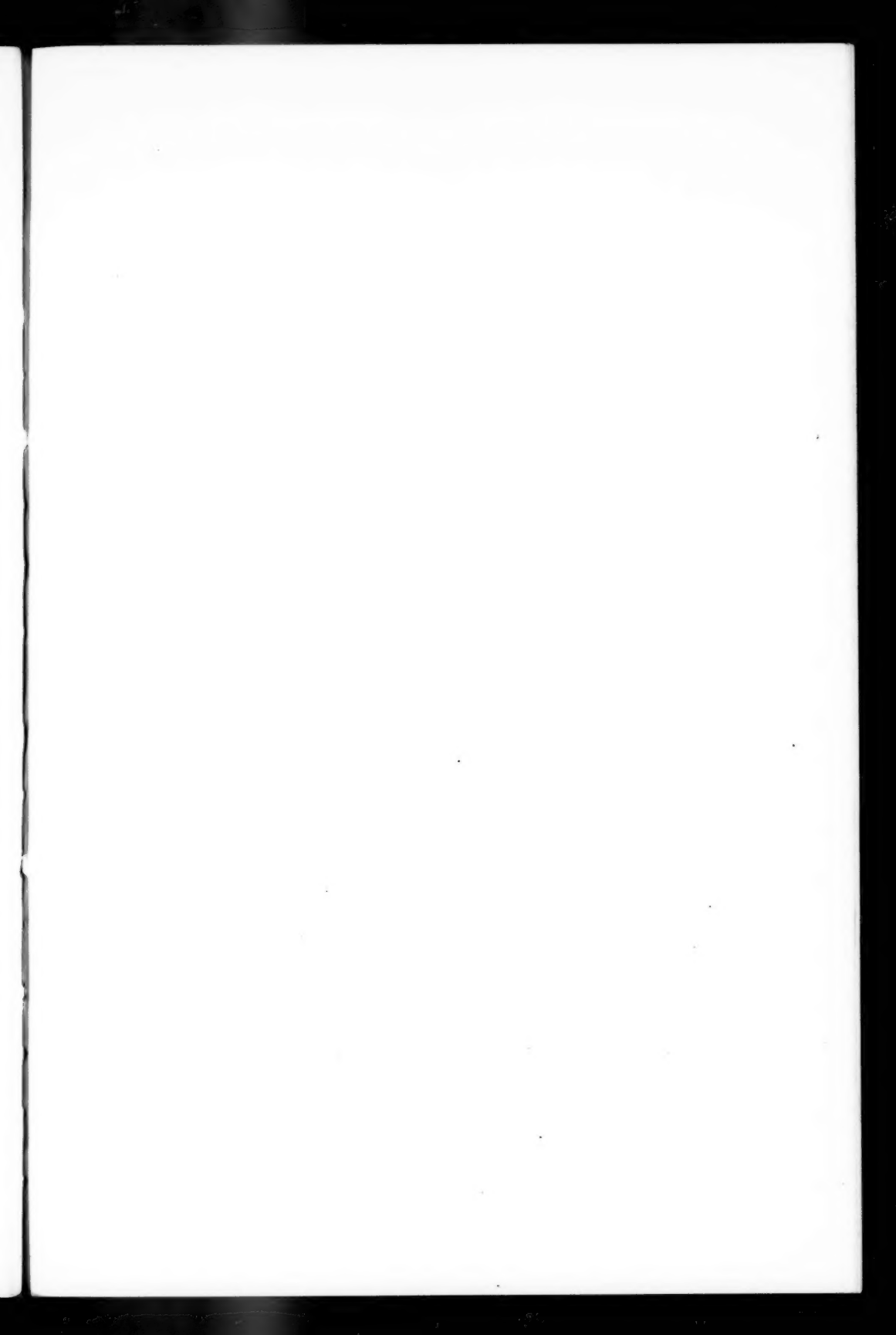
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**EDWARD JENNER.**

(1749-1823.)

Photograph of the painting by Hobday presented to the Royal Society of Medicine  
by Dr. W. S. A. Griffith.

## The Royal Society of Medicine.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

### JENNER CENTENARY.

(January 26, 1923.)

Edward Jenner.<sup>1</sup>

By Sir WILLIAM HALE-WHITE, K.B.E., M.D. (President).

(Consulting Physician to Guy's Hospital.)

. . . . . This is an Art  
Which does mend Nature, change it rather; but  
The Art itself is Nature.

*Winter's Tale*, Act iv, Sc. 4.

A HUNDRED years ago to-day Edward Jenner died. His genius so brightly and so fruitfully illuminated medicine that it seems only right for us to meet to commemorate his centenary, chiefly because the object of this Society is the promotion of both the science and the art of medicine, but also because he was one of our foundation members and contributed two papers to the first volume of our *Transactions*.

He was born in the vicarage at Berkeley in Gloucestershire, on May 17, 1749. He was the third son of Stephen Jenner, the vicar. His mother was the daughter of the Reverend Henry Head who had formerly also been vicar of Berkeley. On both sides he came of an ancient family. Many of his relations were clergymen; his father had been tutor to one of the Earls of Berkeley and, as we shall see, this family always had a great regard for Edward Jenner. Soon after his birth, his father died and he was brought up by his eldest brother, the Reverend Stephen Jenner.

When eight years old he went to school at Wotton-under-Edge but later the Reverend Dr. Washbourn, of Cirencester, undertook his tuition. He was next sent to be an apprentice to Mr. Ludlow, a practitioner in Sodbury; in 1770 he came to London and was for two years a resident pupil in the house of John Hunter. Nothing could have been more fortunate, for both were enthusiastic naturalists, always observing, inquiring and speculating. Before he was aged nine, Jenner had made a collection of the nests of the dormouse; when in Cirencester he collected fossils. Hunter's influence must have greatly stimulated such a mind as Jenner's; they became great friends. On Hunter's recom-

<sup>1</sup> An address delivered at a meeting of the Society held on January 26, 1923, to commemorate the centenary of the death of Edward Jenner on January 26, 1823.



mentation he was appointed to examine and prepare the specimens of natural history brought back by Captain Cook, in 1771, and was offered the post of naturalist to the expedition which sailed in 1772; this he declined.

After Jenner left London many letters passed between them until Hunter's death in 1793. Unfortunately, all Jenner's have been destroyed, but Hunter's have been preserved and, from them, we learn that these two men kept up an extraordinary correspondence. Hunter was always much interested in the hibernation of animals and their temperature, and Jenner made many observations for him especially on bats and hedgehogs. There are many allusions to these animals. In one letter Hunter thus disposes of Jenner's disappointment in love: "I own I was glad when I heard you was married to a woman of fortune; but let her go, never mind her. I shall employ you with hedgehogs, for I do not know how far I may trust mine. I want you to get a hedgehog at the beginning of winter and weigh him; put him in your garden, and let him have some leaves, hay or straw to cover himself with which he will do; then weigh him in the spring and see what he has lost. Secondly I want you to kill one at the beginning of the winter to see how fat he is, and another in the spring to see what he has lost of his fat. Thirdly, when the weather is very cold, and about the month of January I wish you would make a hole in one of their bellies and put the thermometer down into the pelvis and see the height of the mercury." Probably no other disappointed lover has been bid to seek solace in hedgehogs. Another begins: "I received yours with the eel," another thus: "What are you doing? How do the hedgehogs go on?" We get a glimpse of Hunter's menagerie when he writes to say an eagle ate one of his hedgehogs; in a later letter he says he will willingly give three guineas for a bustard; there is a long letter about colour vision and indeed nine-tenths of the letters are taken up with requests for specimens or for experiments to be made in natural history. That Jenner had in him the soul of a naturalist is shown by the fact that one of Hunter's letters begins: "I do not know anyone I would sooner write to than you: I do not know anybody I am so much obliged to." We see further proof of Jenner's capacity as a naturalist when we find Hunter writing to him to suggest that he should come to London and that they should teach natural history.

Jenner was incited by Hunter to study the habits of the cuckoo and on March 13, 1788, there was read before the Royal Society: "Observations on the Natural History of the Cuckoo. By Mr. Edward Jenner. (In a letter to John Hunter, Esq., F.R.S.)" Sir Joseph Banks said that many could hardly believe it wholly. The author tells us that the cuckoo usually lays its egg in the nest of a hedge-sparrow who often turns out her own eggs but never those of the intruder. If any hedge-sparrows are hatched the young cuckoo throws them out of the nest by getting them on its back and jerking them out, its back being especially formed to enable it to do this. Some naturalists have said that the structure of the cuckoo is such that it cannot incubate an egg but Hunter shows that it can. He suggests that the singularities of the cuckoo may be owing to the short residence this bird makes in the country where it is destined to propagate its species, and the call nature has upon it during that short residence, to produce a numerous progeny. All this is especially interesting now, for within the last twelvemonth, cinematograph films have been exhibited showing the young cuckoo throwing out its young companions exactly as Hunter described, and the whole subject of the cuckoo is much to the fore among ornithologists. Beautiful photographs have been taken by Mr. Edgar Chance, the author of "The Cuckoo's Secret," by

Miss Turner and others. Both correspondents were evidently fond of pictures, for Hunter offers to Jenner some of his oil paintings, by George Barrett, the painter in oils, who was the father of the water-colour painter of the same name.

When Jenner returned to Berkeley, after his sojourn with Hunter, he began general medical practice. He took the same interest in this as in other branches of natural history. His talents gained confidence and esteem for him, his practice and his reputation rapidly increased. A patient in the infirmary at Gloucester required an immediate operation, neither of the surgeons were available so Jenner, at Berkeley, sixteen miles off, was summoned; he went and performed the operation successfully. He was disappointed by the uncertainty of action of tartar emetic which he ascribed to imperfections in its preparation. He made experiments to improve this and the process he adopted was published. Hunter writes: "I am puffing off your tartar as the tartar of all tartars. . . . Let it be called Jenner's Tartar Emetic." In 1777, Hunter wrote to Jenner saying he was ill; Jenner suspected he was suffering from angina pectoris, visited him at Bath and found his surmise was correct. He wrote to Heberden to say that, as a result of post-mortem examinations, he had come to the conclusion that angina pectoris was due to disease of the coronary arteries but that he hesitated to make his opinion public or to tell Hunter, for fear of the distress of his friend when he learnt that he had an incurable malady. This is the first account of the association of diseased coronary arteries and angina pectoris. Jenner told his views to his friend, Dr. Parry, of Bath, who regarded Jenner as an excellent pathologist, and recorded them in his book, "*An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris,*" published in 1799, but the author of them had previously expounded them in a paper read before a society of doctors which met at the Fleece Inn, Rodborough, and which he had been instrumental in founding. Jenner, in a letter to Parry, gives the following quaint account of his first case: "I was making a transverse section of the heart pretty near to its base, when my knife struck against something so hard and gritty as to notch it. I well remember looking up at the ceiling, which was old and crumbling, conceiving that some plaster had fallen down. But on further scrutiny the real cause appeared: the coronaries were become bony canals." He also read, before the same society, a communication on heart disease occurring during rheumatism, most unfortunately lost; had this not been so we might perhaps be able to attribute to Jenner the discovery of this important association. Anyhow his paper was original. This society was called the Medico-Convivial in contradistinction to another, the Convivio-Medical, which met at the Ship Inn at Alveston and of which Jenner was also a member. Here he was accustomed to bring forward the reported prophylactic virtues of cow-pox and earnestly to recommend his friends to prosecute the inquiry. His membership of these two societies shows that he was popular with his fellow doctors; his kindness, courtesy and skill certainly—as we know from letters—made him so with his patients. He frequently spent days in the houses of particular friends, especially if any of them were ill, he carrying on his practice from his temporary quarters. Educated people loved his conversation. He used to encourage those whom he liked, to ride twenty or thirty miles with him on his rounds and he would talk to them the whole way. When he left a patient's house often some of the family would ask permission to ride home with him, even if it was midnight. His range of conversation was vast: it was some-

times serious, at others witty. That his command of words was good is shown by the circumstance that he was no mean poet. Miles of far worse poetry than his two "Addresses to a Robin" and his "Signs of Rain" have been printed. He was fond of epigram, thus:—

*On the Death of a Miser.*

Tom at last has laid by his old niggardly forms  
And now gives good dinners; to whom, pray? The worms.

He could sing, and play the violin and flute.

Jenner's practice was so large that in 1783 his nephew Henry became his apprentice. This was no sinecure, for besides purely professional work he had a daily walk of four or five miles to see how the young cuckoos progressed. On March 6, 1788, Jenner married Miss Catherine Kingscote, and on January 24, 1789, his eldest son Edward was born, John Hunter standing as godfather.

In 1792, that is to say when he was 43, the fatigues of an ever increasing practice had become so great that he resolved to practise solely as a physician, and accordingly he obtained the M.D. degree of the University of St. Andrews. He had been a general practitioner for twenty years, during which, in addition to his clinical and pathological professional work, he had carried out observations on the migration of birds, on the cuckoo, and on geology; he had dissected numberless animals, had experimented with hydrogen balloons and had conducted innumerable investigations for Hunter on hedgehogs and a host of other animals.

What sort of man was this unique country practitioner? His friend Gardner describes him thus: "His height was rather under the middle size, his person was robust but active and well formed. In dress he was particularly neat. . . . He was dressed in a blue coat and yellow buttons, buckskins and well polished jockey boots with handsome silver spurs and he carried a smart whip with a silver handle. His hair, after the fashion of the times, was done up in a club and he wore a broad-brimmed hat." Everything we know of him indicates that he was usually cheerful and sociable, very occasionally he was depressed, and we find him complaining that he was an example of the sin of indolence. Many busy people, being aware how much there is to do in the world, at times blame themselves for this.

I have tried shortly to picture to you Jenner the naturalist, pathologist, and country doctor, but now we come to that part of his work which has made him one of the greatest benefactors to the human race. Before his great discovery small-pox was an awful scourge; in 1572 a special medal was struck to commemorate Queen Elizabeth's recovery from it; in the latter half of the eighteenth century it killed forty thousand people in this country every year: many who got well were disfigured for life. Jenner not only showed how to so well prevent this frightful malady that most doctors to-day have never seen a case of small-pox, but from his discovery followed the principle of vaccination which, for example, has in the late war saved whole regiments of soldiers from typhoid fever.

When he was an apprentice at Sodbury a young woman came to his master's house. Small-pox was mentioned. She at once said, "I cannot take that disease for I have had "cow-pox." This remark set Jenner thinking and he pondered on it all his life; he talked about it to Hunter who characteristically told him: "Don't think, but try; be patient; be accurate." When he went

into practice Jenner, as we have seen, discussed the question with other doctors, who were sceptical, for they said cow-pox often did not protect from small-pox. This difficulty Jenner overcame by finding that all the eruptions on the udder of cows were not true cow-pox, and that the vesicles of cow-pox were not equally virulent at all periods of their development. By 1780 he believed he was in sight of a means of completely eradicating small-pox by inoculation with cow-pox, and he was able to say to his friend Gardner, when riding between Gloucester and Bristol: "Gardner, I have entrusted a most important matter to you, which I firmly believe will prove of essential benefit to the human race."

In 1789 he inoculated his eldest child Edward, then 18 months old, with swine-pox matter; subsequently variolous matter was inserted into the child, and again in 1791 and 1792. None of these small-pox inoculations caused small-pox in the child. On May 14, 1796, matter was taken from the hand of Sarah Nelmes, who had been infected with cow-pox by her master's cows, and inserted into the arm of James Phipps, aged 8½. The following July variolous matter taken immediately from a small-pox pustule was carefully inserted by several incisions. Days of the intensest anxiety to Jenner dragged by, but James Phipps did not have small-pox. The experiment up to which Jenner had bent his thoughts for twenty-seven years had succeeded and May 14, 1796, became one of the red letter days in the calendar of every nation on the globe.

He now writes to Gardner:—

DEAR GARDNER,—As I promised to let you know how I proceeded in my inquiry into the nature of that singular disease the cow-pox, and being fully satisfied how much you feel interested in its success, you will be gratified in hearing that I have at length accomplished what I have been so long waiting for, the passing of the vaccine virus from one human being to another by the ordinary mode of inoculation.

A boy named Phipps was inoculated in the arm from a pustule on the hand of a young woman who was infected by her master's cows. Having never seen the disease but in its casual way before, that is when communicated from the cow to the hand of the milker, I was astonished at the close resemblance of the pustules, in some of their stages, to the variolous pustules. But now listen to the most delightful part of my story. The boy has since been inoculated for the small-pox which, as I ventured to predict, produced no effect. I shall now pursue my experiments with redoubled ardour.

Believe me, yours very sincerely,

EDWARD JENNER.

Berkeley, *July 19, 1796.*

Dr. Wollaston hardly exaggerated when he spoke of Jenner as the author of the most valuable communication ever made to the public.

He continued his observations and in 1798 published his famous little book of only seventy-five pages usually called for short the "Inquiry," for the title is very long, as you see by the title-page which I throw on the screen. It contains the record of numerous cases. Some show that persons who have had cow-pox do not get small-pox either when exposed to it or when inoculated with it, some show that if a person has had small-pox he is protected against cow-pox, others show that those inoculated with cow-pox are immune to small-pox inoculations and others that material taken from the cow-pox pustule in the human being may be used to inoculate other human beings who are thus equally protected from small-pox.

He was in London from April 24 till July 14, 1798, trying to persuade persons to be inoculated, but, as the "Inquiry" was not published till the end

of June, people in London knew nothing about the matter and he was unsuccessful, except with Mr. Cline, who inoculated a boy with cow-pox matter given him by Jenner, who had taken it from the pustule of a patient whom he had inoculated with cow-pox. The boy was subsequently inoculated with small-pox but did not have the disease. Cline became an enthusiastic supporter of vaccination. He advised Jenner to leave the country, take a house in Grosvenor Square, prophesying he would become famous and make £10,000 a year. Jenner refused, saying: "Admitting . . . that I obtained both, what stock should I add to my little fund of happiness?" He remained at either Berkeley or Cheltenham till February, 1799. He had now very little time for his practice because he was chiefly occupied with answering the very large number of doctors who wrote to him about vaccination. Many people believed in him, such as Mr. Hicks, who was the first gentleman to submit his own children to the new practice, another early convert was Lady Ducie who had her child vaccinated; the Countess of Berkeley warmly supported Jenner, and, by the next year, Royalty did the same. Most doctors and the best in the profession, such as Abernethy and Lettsom, saw that a great discovery had been made. There were a few who gave trouble, for poor Jenner says, "Brickbats and hostile weapons of every sort are flying thick around me," and, "I am beset on all sides by snarling fellows." He returned to London early in 1799, and this year the "Inquiry" found its way on to the Continent. Dr. De Carro, of Vienna (of whom Richard Bright gives a description in his "Travels from Vienna through Lower Hungary, with some remarks on the state of Vienna during the Congress in the year 1814"), vaccinated his own children, and, by his enthusiastic advocacy, did much to get the practice known throughout Europe: May 14, the day on which Phipps was vaccinated, became an annual festival in Berlin. The French Government recommended vaccination, offering it gratuitously to all who could not pay for it. Thanks to Lettsom, who by his writings did much to help Jenner, it made its way to America. Next year, 1800, he was again in London where he successfully opposed the pretensions of a "Vaccine Board" that would have done more harm than good, and about this time the Bath Vaccine Institution was formed under Jenner's guidance. By 1801 vaccination may be said to have spread all over the world. For example, in Palermo, "It was not unusual to see in the mornings of the public inoculation at the hospital a procession of men, women and children conducted through the streets by a priest carrying a cross, come to be inoculated." The medical officers of the British Navy presented Jenner with a gold medal. He himself offered a thousand guineas towards defraying the cost of getting vaccination to India but he was not called upon for this money as De Carro had already been the means of introducing it there. In Geneva a priest exhorted the members of his congregation to be vaccinated and had a physician present to do it for them after the service. An English clergyman preached a sermon advocating it, and, whenever he baptized a child, gave the parents a tract urging vaccination. The Dowager Empress of Russia recommended it fervently, decreeing that the first child in Russia to be vaccinated should be called Vaccinoff; it was a girl, and provision was settled on her for life. The Empress sent Jenner a diamond ring. A letter from Dr. Davids, of Rotterdam, is addressed to the "Benefactor of Mankind, Dr. Jenner." It is wonderful to see how quickly and ubiquitously vaccination had spread in the three years that had passed since the publication of that little book, the "Inquiry."

By 1802 Jenner's time had been so taken up with going backwards and

forwards to London, with his world-wide correspondence and with gratuitous vaccination that his practice, which was his livelihood, disappeared. His admirers, the Earl and Countess of Berkeley, raised a subscription in his own county of Gloucestershire to present him with a service of plate; but many persons, several of them of great influence, thought that as his discovery saved thousands of lives every year, as the use to be derived from it had spread to the whole civilized world, as he had not concealed it but had given it for the benefit of mankind not attempting to make money out of it, and as his labours had ruined his private practice, he ought to be rewarded by Parliament. Accordingly a petition was presented. This was referred to a committee who examined witnesses and advised a grant, finding that Jenner had reaped no pecuniary advantage from his discovery but that he was a loser by it. On June 2, 1802, Admiral Berkeley in presenting the report of the Committee to the House of Commons spoke of Jenner's discovery as unquestionably the greatest ever made for the preservation of the human species. Other speakers drew attention to his having forgone his livelihood to give his discovery free to the world. Two sums, namely, £20,000 and £10,000, were proposed; by a majority of three £10,000 was given. During this year honours from all over the world were showered upon him. Meetings were held in Paris and Breslau at which a copy of the portrait of him, by J. R. Smith, was wreathed with flowers. The President of the United States wrote to him. He appreciated most, as we all do, the praise of members of his own profession. Medical societies in this country passed resolutions in his honour, but what gave him as great pleasure as anything was that he was invited to address the Physical Society of Guy's Hospital, at a debate on the subject of vaccination. He himself attended on four successive nights. On entering the theatre he was constantly received with universal and rapturous applause. Probably no meeting "ever attracted in a higher degree the attention of professional and scientific men." A new order of merit was instituted by the Society, and Jenner was admitted to it. I am able to throw on the screen the diploma he then received. A pamphlet by John Birch, surgeon to St. Thomas's Hospital, and an opponent of vaccination, is interesting in connexion with Jenner's visit to Guy's. He says:—

"The anniversary of Mr. Guy's Hospital was held in 1802, where I expected to meet the professors, the medical gentlemen and the students on the same terms as usual. What was my surprise, then, to find, that the sole business of the meeting was to begin a canvass for names to a petition to Parliament in support of Dr. Jenner's bill. I refused to sign it. My surprise was increased after dinner to find that toasts and songs and compliments from one professor to another in honour of vaccination were the order of the day."

The Physical Society also presented Jenner with a testimonial, which I, this evening, show you; it is, you will see, signed by the six presidents and 106 members.

In 1803 a meeting, with the Lord Mayor in the chair, was held; as a result the Royal Jennerian Society was founded for "the extermination of the small-pox." The King was patron, other members of the Royal Family were vice-patrons, and Jenner was the first president.

His friends now thought that the rare distinction of a Parliamentary grant would so enhance his reputation that he would make a handsome income if he settled in London; consequently he tried this plan, practising at 14, Hertford Street, Mayfair, but it was a failure. In the first place the grant was not paid



for at least two years and £1,000 was deducted for expenses. Most persons seemed to consider that as he had the grant he was the servant of the public, thus he was inundated with letters and callers; so many were able to vaccinate that few came to Jenner for this, although he was called upon to vaccinate numbers of patients gratuitously. Therefore, after giving practice in London a fair trial, he relinquished it, returned to Berkeley and again became the village doctor, coming to London very rarely. But the experiment had been extremely costly, and consequently his supporters brought before Parliament the question of a further grant. The Government called upon the Royal College of Physicians to report upon the subject of vaccination. This body examined the matter most carefully and judiciously, and on April 10, 1807, presented a report signed by Sir Lucas Pepys, the president. It strongly recommended the practice of vaccination, the truth of which "seems to be established as firmly as the nature of such a question admits," and the College considered that the public may look forward to the end of the ravages, if not the existence, of small-pox. As a result of this report the Chancellor of the Exchequer proposed a grant of £10,000. Mr. Edward Morris, the member for Newport in Cornwall, moved an amendment to make it £20,000. The amendment was carried by a majority of thirteen with, for Jenner, the fortunate condition attached that the grant was to be free of fees.

He now lived at his house "The Chantry" in Berkeley, doing the ordinary work of a country practitioner. In the garden was an arbour in which he used to vaccinate the poor gratuitously. Sometimes he visited Cheltenham or Bath, occasionally he had the opportunity of seeing celebrated visitors to his neighbourhood. He delighted in walks and rides; he was as eager as ever in the study of nature. There exists a charming note by him describing how moths feed upon the night-blowing primrose. It was now that he did most of his work on the migration of birds, but his enormous correspondence must have occupied much of his time. In no wise was he puffed up with his fame, he remained a simple unostentatious man, a friend of his neighbours whom he liked to meet at dinner. He gave freely of that most valuable of all commodities, his time; he was assiduous as a magistrate; he would listen to all callers, rich or poor, at whatever time they came to him. He was particularly cordial to all his fellow practitioners, and especially liked talking with the younger of them. His kindness to the poor was continual. Phipps became very ill, therefore, as he lived in a miserable place, Jenner built him a comfortable cottage, himself stocking the garden with shrubs and flowers. One of the villagers showed considerable musical ability, whereupon Jenner took him to a music meeting at Gloucester. Indeed, as far as we can gather, he led the happy life of the perfect country doctor.

Nevertheless, in the last twenty years of his life, which he spent almost entirely in the seclusion of a country village, his fame continued to spread and honours fell thick upon him from all over the world. The list of diplomas, honours and addresses awarded to him numbered, according to Baron, forty-seven and of these more than half were received after his final return to the country. The freedom of the City of London was presented to him in a box valued at a hundred guineas. He was also made a freeman of Edinburgh, Glasgow, Dublin, Liverpool and Kirkcaldy. He received an honorary degree from the University of Oxford, he was made physician extraordinary to the King. One of the greatest of his distinctions was that he was unanimously elected a Corresponding Member of the National Institute of France, and later on he was made one of its Foreign Associates. Addresses, resolutions,

letters from societies and from distinguished people, not included in the above forty-seven, all thanking the discoverer of vaccination were almost innumerable. No country welcomed vaccination more warmly than Spain. The Government fitted out an expedition to carry the practice to all Spanish dependencies throughout the world. It was under the care of Dr. Balmis, and set sail from Corunna on November 30, 1803. There were on board twenty-two children who had never had small-pox, selected for the preservation of the vaccine fluid, by transmitting it successfully from one to another during the voyage. In three years the expedition returned to Spain, having circumnavigated the globe and successfully introduced vaccination to Central America, South America, and many other places.

It was carried even to the natives of North America, who welcomed it. Jenner sent them his book. The chiefs held a meeting to thank him, and in their reply said:—

“We shall not fail to teach our children to speak the name of Jenner; and to thank the Great Spirit for bestowing upon him so much wisdom and so much benevolence. We send with this a belt and a string of Wampum in token of our acceptance of your precious gift; and we beseech the Great Spirit to take care of you in this world and in the land of Spirits.”

Then follow the native signatures and marks which I show you.

The European inhabitants of India gave Jenner over £6,000 as a testimony of their gratitude. Vaccination spread to the most remote parts of Russia and China; in this last country a book about it was published. In Italy, Dr. Sacco and his assistants vaccinated 1,300,000 persons in eight years. Coleridge projected a poem on vaccination; an Italian poem of 286 pages, “The Triumph of Vaccination,” was published. During his last visit to London Jenner had several interviews with the Duchess of Oldenburg, sister of the Emperor of Russia, and one with the Emperor himself.

Such was the gratitude of mankind to him that he attained an influence throughout the world probably never before or since acquired by a private individual. Dr. Wickham and Mr. Williams were detained in Geneva by Napoleon. All efforts to obtain their release had been fruitless. Jenner wrote to Napoleon asking that they should be set free, and they were, Napoleon saying: “Jenner! Ah, we can refuse nothing to that man.” On another occasion he liberated two other Englishmen on Jenner’s request. The Emperor of Austria also set free an Englishman when asked by Jenner, and on his intercession the King of Spain gave a prisoner his liberty. Those who travelled abroad were able to dispense with passports if they took a certificate by Jenner, for it was respected everywhere. Here is an example:—

“I hereby certify that Mr. A., the young gentleman who is the bearer of this, and who is about to sail from the port of Bristol on board the *Adventure*, Captain Vezey, for the island of Madeira, has no other object in view than the recovery of his health.

EDWARD JENNER,

Member of the N.I. of France.

Berkeley, Gloucestershire, July 1, 1810.”

In his letter to the young man’s father Jenner says that if the boy is captured the certificate will secure his release.

Jenner’s portrait was painted by Lawrence; one by Northcote is in the National Portrait Gallery. That by Hobday is considered the best, and I am



happily able to show it to you, for it has been presented to this Society by the great generosity of Dr. W. S. A. Griffith. His portrait was also painted by J. R. Smith. Many medals have been struck in his honour. Statues have been put up to him in many countries; the London statue is in Kensington Palace Gardens. Collections of things of interest about him have been made. Probably the best is that in the Wellcome Historical Medical Museum, where a special exhibition is being held in honour of this centenary. Thanks to the kindness of Mr. C. J. S. Thompson many objects from that museum are being exhibited here to-night, and I have to thank him for several photographs. Institutes have been called after Jenner. Centenaries were held to celebrate the first vaccination. Paris, like London, is holding a centenary to commemorate Jenner's death. To-day a wreath has been placed at the foot of his statue in Gloucester Cathedral, and an "In Memoriam" notice has appeared in the *Times*.

When you look at Hobday's portrait you will not be surprised to hear that Jenner was liable to apoplexy. His first attack was on August 6, 1820, from this he recovered completely; his final and fatal attack was a hundred years ago to-day.

Usually when a really great discovery is made it is attacked by ignoramuses, fools, knaves and cranks. In their day they had their fling at Jenner, but I have not alluded to them, for time and Jenner's fame have cast them into obscurity, where they had better stay. Nor have I given any proofs of the efficacy of vaccination; this has been done over and over again, and before such an audience as this is quite unnecessary. Rather have I tried to delineate the quiet, retiring country doctor, fond of music, poetry and painting, beloved by those who knew him, quite unspoilt by fame such as has fallen to but very few, a great naturalist, the friend of Hunter, a man who would have been known as a pioneer pathologist had he published his observations, one who by patient thought and observation extending over many years, arrived at a discovery which has helped us to banish one of the greatest scourges of mankind, the principle of which has since been applied equally successfully to other diseases, and has been named vaccination by Pasteur in honour of Jenner.

"And he stood between the dead and the living and the plague was stayed."—*Numbers* xvi, 48.

## The Royal Society of Medicine.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### PASTEUR CENTENARY.<sup>1</sup>

#### Pasteur in Relation to Medicine.

By Sir WILLIAM HALE-WHITE, K.B.E., M.D. (President).

*(Consulting Physician to Guy's Hospital.)*

A FEW weeks ago was the Centenary of the birth of Louis Pasteur, for he was born on December 27, 1822, at Dôle. The Royal Society of Medicine could not pass by such a date, hence this meeting.

It was a remarkable time in the history of medicine. Jenner whose fame was world wide, was still alive. Laennec, that prince of observers, had, two years before, published "*L'Auscultation Médiate*" and the thin bony man, already marked for death by phthisis, began his first course of lectures at the Royal College of France three weeks before Pasteur's birthday. Bright, who became the best known British physician since Harvey, had settled down at Guy's Hospital, where he made his immortal observations.

All these four were for a month alive at the same time. The three elders owe their celebrity to having had, to an exalted degree, the faculty of observing the experiments made by nature. The infant just born attained his by observing the experiments he himself made; he broke newer ground and reached wonderful results, but all four were indomitable workers, marvellously accurate observers and with them all mere doctrines must give way to the truth.

Pasteur's father was a tanner in a small way of business who had, in the Napoleonic wars, been created a Chevalier of the Legion of Honour on the battlefield. He was fond of reading and had much influence on Louis, who said on the death of his father: "For the last thirty years I have been his first and almost his only interest in life. I owe everything to him." The old soldier was intensely patriotic and his continual talk of his dear country impregnated his son with ardent patriotism, which led him in 1871 to return to the University of Bonn the diploma of the honorary degree of M.D. saying: "The sight of this parchment is now hateful to me, and I feel it an insult to have my name . . . associated with one doomed from henceforth to the execration of my fellow countrymen, that of Rex Gulielmus." Likewise in 1895 he refused the Prussian Order of Merit.

The world owes much to Pasteur's wife. He was appointed Professor of

<sup>1</sup> Celebrated by the Society, February 28, 1923, by a "Social Evening," attended by His Excellency the French Ambassador (the Count de Saint-Aulaire), and many distinguished guests.

Chemistry at the University of Strasbourg in January, 1849, and within a fortnight had fallen in love with the daughter of the Rector, M. Laurent, and he married her on May 29 of the same year. During the whole of their married life, she appreciated that she had become the helpmate of a great genius. When he was working on a problem he became oblivious to the outside world. He forgets that he has promised to take her to an important festivity; she waits hours, he does not come; but there is no reproach from her. On occasions she has either to send or to fetch him home from the laboratory to his meals, if not he works on for hours forgetful of time, she shields him from all domestic affairs, never allows, if she can prevent it, worries to intrude upon him, she is never jealous of his devotion to science, in the evening she takes down notes of the day's work from his dictation, helps him to state things clearly by asking him for explanations; not only was she an incomparable companion but his best collaborator. That he would have given less to humanity had he married a different wife must be true. The Gods smiled on us all when he married Madame Pasteur.

In childhood he showed no particular promise except as a draughtsman—about this Dr. Monod will tell us—as a young man he became deeply and enthusiastically interested in chemistry. His work in this Professor Lowry will describe.

He was first led to micro-organisms because the common mould, *Penicillium glaucum* grows in a liquid containing paratartaric acid, but consumes only the dextro-rotatory tartaric acid leaving the lævulo-rotatory tartaric acid untouched. Pasteur went from Strasbourg to be Professor and Dean of the Faculty of Science at Lille, where alcohol was manufactured from beetroot by fermentation. The father of one of his pupils was in difficulties because, for reasons that he could not see, sometimes the process turned out badly. Up to this time fermentation was unexplained. Pasteur determined to get to the bottom of it and showed that the formation of alcohol from sugar was a vital process connected with the growth of a specific micro-organism which performed the conversion, and that in the same way another specific micro-organism had the property of forming lactic acid from sugar. He left Lille and came to the École Normale in Paris where his laboratory consisted of two attics under the roof, overpoweringly hot in summer, freezing cold in winter, and he had no assistant of any kind. The studies on fermentation were continued and he showed for the first time that butyric acid fermentation, numbers of diseases of wine, vinegar and beer, were all due to micro-organisms which he divided into two great classes, aërobes requiring free oxygen to maintain their life and anaërobes capable of living without free oxygen but able to wrest this element from its combination with other elements. Each fermentation, each disease had its own specific micro-organism and all could be prevented if living micro-organisms were excluded. Now that all this is common knowledge it is difficult to put ourselves back fifty years when it was not, but if we do, and then reflect, we dimly appreciate what a revelation Pasteur made.

His friend Dumas, the chemist, who from the first saw that Pasteur was a genius, begged him to study silkworm diseases which were then causing the loss of immense sums of money and leaving thousands of people without the means of a livelihood. Pasteur said he had never touched a silkworm, but he consented and went to live in the district, spent five or six years on the subject, showed that there were two diseases, that each was due to a micro-organism and each was communicable from worm to worm just as typhoid fever is communicable from man to man. Having found the cause it was easy

for Pasteur to show how to prevent the malady and the large poverty-stricken and famine-stricken districts were restored to prosperity.

When investigating chicken cholera, a disease of fowls due to a micro-organism, Pasteur made the astounding discovery that if these micro-organisms were left for a long while in the laboratory to live on a suitable soil they gradually lost their excessive virulence because of long exposure to the oxygen of the air. If, now, fowls were inoculated with these micro-organisms of weakened or attenuated virulence, the birds did not die but were only slightly ill, but if, subsequently, they were inoculated with virulent chicken cholera micro-organisms, they showed no signs of disease although fowls inoculated with the same micro-organism, but not previously inoculated with the attenuated virus, quickly died. He had succeeded in vaccinating fowls against chicken cholera, and had made the grand discovery that it was possible to attenuate the virulence of a micro-organism outside the body, and that inoculation with the attenuated virus protected against the disease due to this micro-organism. This doctrine of attenuation of the virus is of fundamental importance in bacteriology and preventive medicine, and its discovery was one of Pasteur's greatest works.

The disease called anthrax, splenic fever or charbon, was the cause of the loss of thousands of sheep and cows. It is due to the anthrax bacillus, which in animals flourishes in the living blood and forms spores which retain their power of growing into active anthrax bacilli for an indefinite time. Many sheep and cows feeding in particular fields died of anthrax. Pasteur showed that this was because animals dead of anthrax had been buried there; worms brought up to the surface spores from the dead animals, these were eaten by sheep and cows whilst grazing, and in them the spores developed into anthrax bacilli which killed them. By growing anthrax bacilli in the laboratory on a suitable soil at a raised temperature he lessened the virulence of these bacilli—attenuated it. Spores from these attenuated bacilli could be given to animals without killing them, but thereby they were protected from anthrax. In other words he discovered that he could vaccinate sheep and cows against anthrax. To prove this, fifty sheep were taken, twenty-five were vaccinated, twenty-five were not. Each twenty-five were kept separate. Twenty-five days later all the fifty sheep were inoculated with virulent anthrax bacilli. The twenty-five vaccinated sheep all remained perfectly healthy; the twenty-five unvaccinated all died from anthrax. Similar successful experiments were performed on cows. The Grand Cordon of the Legion of Honour was given to Pasteur and the Ribbon of the Chevalier to his assistants Chamberland and Roux.

Swine fever, or rouget, is a terrible disease. In the United States in 1879 it killed 900,000 pigs. Pasteur discovered a vaccine, that is, an attenuated virus for this, but attenuation was produced in a new way. He found that the micro-organism of this disease was fatal to pigs, pigeons and rabbits. If it was transmitted from pigeon to pigeon it rapidly and greatly increased in virulence; if from rabbit to rabbit it decreased in virulence, so that by such passage an attenuated virus might be prepared, and this he successfully used to vaccinate pigs against swine fever.

If we consider Pasteur's work on diseases of wine, beer, vinegar, silkworms, chicken cholera, anthrax and swine fever we see the truth of Huxley's remark: "Pasteur's discoveries alone would suffice to cover the war indemnity of five milliards paid by France to Germany in 1870."

Soon after Pasteur began the study of micro-organisms he saw that his work must bear on medicine, for in 1863, in an interview with the Emperor,

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he assured him that all his ambition was to arrive at the knowledge of putrid and contagious diseases. He pondered on the words of Robert Boyle who, about 200 years before, had said: He that thoroughly understands the nature of ferments and fermentations shall probably be much better able, than he that ignores them, to give a fair account of divers phenomena of certain diseases (as well fevers as others), which will perhaps be never properly understood without an insight into the doctrine of fermentations." Pasteur's work on fermentation led him to the conclusion that putrefaction, like fermentation, is due to micro-organisms which are usually derived from the air. Quite early he showed that when urine decomposes to form ammonia, a special micro-organism is at work, that its activity can be inhibited by boracic acid, consequently this acid was successfully used in medicine for this purpose.

He had not any medical qualification, nor any clinical training, nevertheless to his great delight he was in 1873 elected by a majority of one vote to the Academy of Medicine. He was a constant attendant, always urging the



Souvenir medal of the centenary of Pasteur's birth (slightly enlarged).—The medal is issued by the Association for the Extension of Pastorian Studies (11, Rue Anatole-de-la-Forge, Paris, 17). The portrait was selected by Madame Vallery-Radot (Pasteur's daughter) as the most faithful likeness of her father. The object of the Association is to encourage well qualified men and women to take up biological research in relation to medicine, agriculture and industry.

importance of micro-organisms in disease, always fighting the many old-fashioned members who denied this. What with his demolition of their opinions and the enthusiasm for the new doctrine with which he inspired the younger members, such as Roux, his membership of this Academy was a happy event for medical science. The contemptuous remarks with which he was assailed show an almost unbelievable denseness of perception. One surgeon alluding to Pasteur's work spoke of "laboratory surgery, which has destroyed very many animals and saved very few human beings."

But the greatest of all the triumphs that have followed from his investigations was, unknown to him at the time, evolved in this country. The mortality due to putrefaction of wounds, whether accidental or inflicted by surgery, was appalling. But when Lister learnt that Pasteur had shown that putrefaction was a fermentation caused by the growth of microbes, and that these could not arise *de novo* in the putrescible substance, a solution of the problem appeared. He revolutionized surgery by proving that putrefaction

after operations could be abolished if the patient's skin, the surgeon's hands, the sponges, dressings and instruments were all so treated that they had no living micro-organisms attached to them and that if micro-organisms were already there, as they often are after injury, the wound would heal without the onset of blood poisoning if they were rendered inert or removed. Nothing affected Pasteur's feelings more deeply than this beneficial result of his teaching. At the meeting of the International Medical Congress held at Copenhagen in 1884, Lister, addressing Pasteur, said: "Truly there does not exist in the whole world an individual to whom Medical Science owes more than to you. . . . Thanks to you, surgery has undergone a complete revolution which has deprived it of all its terrors and has increased its efficacy to an almost unlimited extent."

Hydrophobia, or rabies, is communicated by dogs or wolves to man by biting. The suffering is dreadful. I have seen people die of it, and can testify that the illness is the most horrible of those with which mankind can be afflicted; untreated it is fatal. The micro-organism is unknown, but Pasteur showed that the virus resides in the nervous system, by injecting an emulsion of the spinal cord of a mad dog into a healthy dog and thereby causing rabies in it which did not show itself for fourteen days. This then is the incubation period. He transmitted the disease by inoculation to a rabbit, and from rabbit to rabbit until the ninetyeth rabbit was reached, and found that, by this means, the incubation period is reduced to seven days. By suspending in air the spinal cord of a rabbit dead of rabies having this short incubation period, he was able, because of the oxygen in the air, to attenuate the virus so that when an emulsion of such a spinal cord was injected into a dog the animal did not suffer from rabies, nor did it get rabies when inoculated with a virulent spinal cord from a mad dog. In other words, the healthy dog had been successfully vaccinated against rabies. In man the incubation period is long. Pasteur inoculated human beings recently bitten by a mad dog with a rabbit's spinal cord containing the attenuated virus, having the short seven days' incubation period, and found to his intense delight that they did not get rabies. By this treatment, he said, we can prevent rabies in human beings bitten by mad dogs and wolves if we get them for treatment sufficiently soon after the bite. An English Commission appointed by Government to investigate the matter reported that he had proved his claim.

When Pasteur was asked to what he owed his success, he said it was to assiduous work, with no special gift but that of perseverance. He was a tremendous worker. I have not mentioned the immense labour he spent in refuting the doctrine of spontaneous generation. But he was much more than a worker. He was an enthusiast. When he gave up chemistry he wrote: "I, who did so love my crystals." He rushed about the country in pursuit of his investigations. At the age of 46 he had a severe cerebral hæmorrhage, but doctors' directions that he must work less made no impression; he never attended social gatherings or theatres; he fought all who disagreed with him; his friends thought he wasted time on this, but, as Tyndall said: "Pasteur is combustible, and contradiction readily stirs him into flame." This was not because he desired any personal glorification, he was most modest, but because he must at all costs uphold what he knew to be correct. He was a marvellous experimenter, proving his assertions up to the hilt by simple but uncontrovertible experiments in which he had guarded against every possible error. He was an implacable



critic of his own and other people's experiments. He had the infinite capacity for taking pains, and an intuition for devising just the right experiment. If an analogy suggested itself no use was ever made of it until it had been submitted to experimental verification; but facts fell upon his mind like seeds on a fertile ground. He had a wonderful faculty for discerning the relative importance of them. He was fond of saying that: "In the fields of observation, chance only favours the mind which is prepared."

Then, too, he had the magnetism which made others love to work with him and follow his inspirations; he did so much that if it had not been for helpers much must have been left undone. He was a genius, a seer and a prophet, always adding to knowledge, always looking forward to more additions. "Science," he said, "has no nationality, because knowledge is the patrimony of humanity, the torch which gives light to the world. Science should be the highest personification of nationality because, of all nations, that one will always be foremost which shall be first to progress by the labours of thought and of intelligence."

Surely what I have said shows that the immortal Pasteur was one of the greatest solvers of nature's secrets the world has ever known, and that the debt owed to him by humanity is unpayable and incalculable.

### Pasteur as Chemist.

By Professor T. M. LOWRY, F.R.S.

IN the brief time at my disposal I shall attempt only two things: first, to present in outline a picture of the work which won for Pasteur the chemist a place in the ranks of the Immortals; second, to indicate the bridge by which his chemical work is linked to those biological researches which enabled a mere chemist to claim the attention of a society such as that which I have the honour to address. My task has been made easy by Pasteur himself, since rather more than sixty years ago he delivered to the Chemical Society of Paris two lectures which cover precisely the same field as that into which I propose to guide you to-night.

Pasteur claimed that in his work on tartaric acid he was following in the footsteps of three physicists—Malus, Arago, and Biot. Malus, in 1808, had announced the discovery of the polarization of light by reflection—that is to say, that light reflected from a sheet of glass was altered in such a way that the vibrations in one plane entered the glass, whilst the vibrations in the perpendicular plane were reflected from it. The plane which contained the incident and reflected ray was described as the *plane of polarization*. Arago, in 1811, discovered that when a ray of polarized light was passed through a plate of quartz the plane of polarization was distorted, giving rise to beautiful chromatic effects. Biot, in 1812, made the further discovery that this distortion took the form of a uniform rotation of the plane of polarization, but that some plates of quartz rotated this plane to the right and others to the left. He also discovered, in 1815, that a similar rotation of the plane of polarization was produced by many natural products, such as oil of turpentine and solutions of sugar, of camphor, or of tartaric acid.

[February 28, 1923.]

We now pass on to consider the relation between the geometrical form of the crystal and its ability to rotate the plane of polarized light. A typical crystal of quartz consists of a hexagonal prism capped by hexagonal pyramids. Haüy, however, detected the presence of tiny facets on the alternate corners, which made the crystals lopsided or asymmetric; he also found that these hemihedral facets could be distributed in two ways, giving rise to crystals which could not be superposed upon one another, although they could be converted into one another by reflection in a mirror. Quartz crystals could, therefore, assume two different geometrical forms, whilst plates of quartz could rotate the plane of polarization of light in two opposite ways. It was left to Sir John Herschel, in 1820, to discover the correlation between these facts, and to show that crystals having one geometrical form always rotated the plane of polarization to the right, whilst those of opposite form rotated it to the left.

I now pass on to consider Pasteur's discovery of similar phenomena in tartaric acid. This acid was known in two forms—the ordinary dextro-tartaric acid, which rotated the plane of polarization to the right, and a modification (which Pasteur called *para*-tartaric acid), which possessed identical chemical

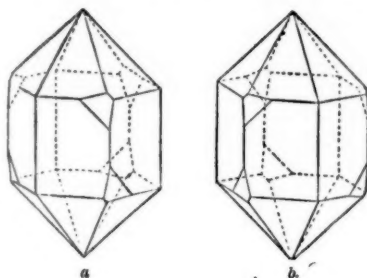


FIG. 1.—(a) Dextro-rotatory quartz; (b) Laevo-rotatory quartz.

properties, but was devoid of optical rotatory power. Pasteur found that tartaric acid and its salts all gave asymmetric crystals, like quartz, whilst the *para*-tartrates gave symmetrical crystals. It appeared, therefore, that there might exist a correlation between the asymmetric crystals of the tartrates and the optical rotatory power of their solutions, similar to that which existed between the asymmetric crystals of quartz and the optical activity of solid plates cut from the crystal.

At this stage Pasteur's attention became fixed upon an observation of Mitscherlich's to the effect that one of the salts of *para*-tartaric acid gave rise to crystals of precisely the same type as the corresponding tartrates. When Pasteur investigated this anomaly he found that, contrary to all previous experience, hemihedral facets appeared on the crystals of the optically inactive *para*-tartrate as well as of its dextro-rotatory isomer. Further examination of this anomaly showed, however, that whereas in the dextro-rotatory tartrate the hemihedral facets were always of one kind, in the *para*-tartrate both types of crystals were formed side by side from the same solution. It appeared, therefore, that when this particular salt was crystallized the *para*-tartaric acid resolved itself spontaneously into two opposite forms of tartaric acid, one of



which had been known for nearly a century, whilst the other, its mirror image, was new to science. It is of interest to notice that in no other known case does this spontaneous resolution of an inactive tartrate take place, and that even the two sodium ammonium salts unite to form a double salt above  $26^{\circ}\text{C}$ . It is, therefore, quite possible that Pasteur's life work might have taken a totally different course if he had begun his work in a tropical instead of in a temperate climate.

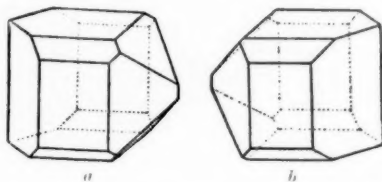


FIG. 2.—(a) Dextro-tartaric acid; (b) Laevo-tartaric acid.

Pasteur was a young student at the time when he made this important discovery, and, before communicating the results to the Academy, Biot insisted that Pasteur must come to him and repeat before his own eyes the decisive experiment. "He provided me," says Pasteur, "with some *para*-tartaric acid, which he had already studied with particular care and which he had found to be perfectly neutral towards polarized light. I prepared the double salt in his presence, using for the purpose soda and ammonia, which he had also wished to provide for me himself. The liquor was left in one of his rooms to evaporate slowly, and, when it had furnished 30 or 40 grm. of crystals, Biot asked me to

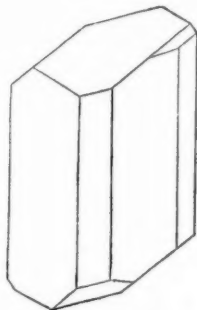


FIG. 3.—Racemic acid.

come to the Collège de France in order to collect them and to separate them under his eyes by their crystallographic character into right and left crystals. He also asked me to state again whether I could assert that the crystals which I placed to his right would rotate the plane of polarization to the right and the others to the left. That done, he told me that he would do the rest. He prepared the solutions of carefully regulated strengths, and, at the moment when he was about to observe them in the polarimeter, he invited me afresh to come into his room. He placed in the apparatus first the most interesting

solution, that which would rotate the light to the left. Without stopping even to make a measurement, he saw, by a single glance at the colours of the ordinary and extraordinary images in the analyser, that there was a strong deviation to the left. Then, visibly moved, the illustrious old man grasped my arm and said to me, 'Mon cher enfant, j'ai tant aimé les sciences dans ma vie que cela me fait battre le cœur.'

The optical rotatory power of quartz is due, as Fresnel had suggested, to a spiral packing of the molecules, and by the kindness of Sir William Bragg I am able to exhibit a model in which the spiral distribution of the atoms of silicon is clearly shown. This spiral structure can be built up in two opposite ways, but when it is destroyed (for example, by melting the quartz) the optical rotatory power vanishes. The asymmetry, then, is in the crystal, but not in the molecules of silica from which it is built up. Tartaric acid, however, displays its optical activity in solution, and we owe to Pasteur the momentous declaration that the *molecules* of tartaric acid, as well as the crystals, must be asymmetric. Here, again, through the kindness of Sir William Bragg, I can show you a model which illustrates both the spiral arrangement of the carbon atoms in tartaric acid and the way in which these spiral molecules are packed together in an asymmetric crystal.

Just a word in reference to the biological applications of these discoveries. Pasteur called attention to the fact that in the mineral kingdom, and by the artificial operations of the chemical laboratory, asymmetric molecules are always produced in equal quantities of opposite types, giving rise to optically inactive compounds or mixtures. Nature, however, almost invariably produces optically active products; and all the materials of the living tissue are branded with this symbol of their divine origin. This, then, is the real distinction between the organic and the inorganic, between nature and man, and this "middle wall of partition" Pasteur was the first to break down. It is indeed a remarkable fact that the three methods which to-day enable us to accomplish the final stage in the synthesis of natural compounds, by endowing our synthetic products with optical activity, were all described by Pasteur more than sixty years ago.

One other link between Pasteur's delicate chemical studies and biology may be mentioned. The mould which ferments ordinary tartaric acid has but little action upon its optical isomer. It was therefore possible by fermenting *para*-tartaric acid to destroy the common dextro-rotatory form, whilst preserving the *laevo*-rotatory isomer. The yeast, trained for countless generations to assimilate dextro-tartaric acid, refuses to ferment *laevo*-tartaric acid, and turns away with loathing from the unaccustomed food. This phenomenon has been widely extended, and applies not only to humble organisms but to man himself. Thus, whilst one form of asparagin tastes sweet in the mouth, the mirror image has an insipid taste. If, therefore, through some freak of nature the asymmetry of the vegetable kingdom were reversed, whilst leaving that of the animal kingdom unchanged, we too, like the yeast, might starve in the midst of plenty, unable to digest the unaccustomed sugar with its facets at the wrong corners, or to nourish ourselves upon the finest wheat flour containing starch and gluten of the wrong sign.

This, surely, is the real tragedy of "Alice through the Looking-Glass." The buns and cakes beyond the mirror would present a tempting appearance; the toffee and the barley sugar would still attract her; but if she should stretch out greedy hands and grasp them through the mirror, she would be doomed to disappointment. The fats she would be able to digest, and it might still be

necessary to warn her to leave the wine decanter alone, since the alcohol has a symmetrical molecule, and would be equally potent from whichever side of the mirror it was derived; but the carbohydrates and proteins would probably "turn to ashes" in her mouth and provide her with little or no nourishment. Even if she should succeed, however, in finding sufficient food, her growth would certainly be arrested by lack of optically active vitamins of the right sign, and death would certainly await her, perhaps the more merciful if not long delayed. Would insulin be of any value to her in the terrible state into which she would inevitably lapse as a result of wandering to the wrong side of the looking-glass? Even the seven happy years that I spent at Guy's have not qualified me to attempt a prognosis, even when as in this case my diagnosis must be accepted as correct. But, greatly daring, I will assert that all the efforts of the Medical Research Council would fail to save Alice, for the remedies that they would prepare on this side of the looking-glass would have no potency in enabling her to assimilate the strange food on the other side of the glass. Alice must begin all over again, and beyond the mirror organize a medical research council of her own. We should recognize them easily, for the wise inhabitants of that other world would know that they could not do better than to reflect "our" choice, and confirm "our" nomination (I speak on behalf of all the inhabitants, both fauna and flora, of our side of the mirror). But they would have some strange habits. Their hair would be parted on the wrong side; they would carry their watches in their right-hand pockets and listen to the beating of hearts on the right side of their patients. In their moments of leisure they would play golf with clubs of an unfamiliar type, with which they would make magnificent left-hand drives; but their handicaps and their feelings would remain the same, since these things are not altered by reflection in a mirror. They would then manufacture from materials gathered on the other side, suitable and potent remedies for the diseases of that other world; and they would apply these to Alice. At last she would look forward to a cure, but only to meet with fresh disappointment, since the cure would prove to be only a treatment of her disease; for after all Alice herself must undergo optical inversion, limb by limb and molecule by molecule, turning her right hand into a left and reversing the sign of her tissues, as her body passed through the plane of the mirror, before she could fit harmoniously into the framework of the universe beyond. And there at last we may leave her, happy and contented, and enjoying all the good things that life "through the looking-glass" has to offer.

Such, briefly, is the concept which Pasteur places before us as his earliest contribution to natural knowledge; and we cannot but admire the perfect fashioning of the foundation stone upon which in his riper years he built up the splendid monument, which generations still unborn will visit, in order to lay before it a tribute of laurel leaves such as we bear in our hands to-night.

## Pasteur as Artist.

By GUSTAVE MONOD, M.D.

I AM representing to-night the "Association pour le Développement des Relations Médicales," of the Faculty of Paris, and I convey to you the friendly greetings of its President, Professor Hartmann, and members, whose pleasure it is to join with you in the demonstration of homage and gratitude to Pasteur.

*Pasteur the Artist.*—Great personalities may be studied under different aspects, and we might consider Pasteur under those of the pioneer, the professor, the polemist, the patriot, the believer, or what he was to such an extent, the family man. But I must limit myself to my title.

I want to show you Pasteur, not only as an artist, in the accepted term, that is one "who bears in his heart an ideal of beauty and who follows it," (remember these words, we shall hear them again), but as an artist in the Latin sense of the word "artifex"; a man who expresses this ideal in his work.

From his childhood he knew how to represent what he saw, and later on his discovery of the *microcosm* was the work of an experimenter who observed with exceptional acuteness and whose imagination was as honest as it was bold.

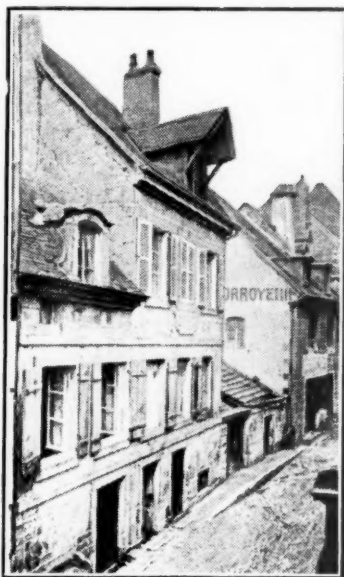
I have brought for exhibition reproductions of twenty drawings by Pasteur. These were published in 1910—fifty collections—and although excellent, they hardly convey the impression of the originals. I have seen these pastels recently; they are owned by his son-in-law, M. Vallery-Radot, author of the *Life of Pasteur*, the "*Histoire d'un Savant par un Ignorant*."

M. Vallery-Radot has reconstituted the home of Pasteur in his mansion at Versailles. We see his furniture, his work-table, his book and the pictures he loved best; amongst them the photographs of his friend Sir James Paget, portraits of him as a young man, and most touching of all a drawing of the calm face of the great man in his last sleep. But what touched me most of all in his sanctuary was the presence of Pasteur's own daughter, with her noble features so strikingly like her father's, reflecting the glory and the tenderness which illuminated her childhood. In this private museum the whole life of Pasteur is told in pictures.

Arbois is a little town of eastern France at the foot of the Jura; its hills and clear, rapid streams recall the Highlands of Scotland. If we imagine ourselves back in 1831, we can picture a band of noisy children coming home along the river bank from fishing. One of them, a boy small for his 13 years, attracts attention by his thoughtful expression, and the passers-by smile, saying, "This is Louis, the son of M. Pasteur, the tanner, our young artist." For Louis Pasteur, without being in any way an infant prodigy, early distinguished himself by his aptitude in drawing. Doubtless, if we possessed his boyhood's exercise books, we should find in the margins more or less unskilful sketches of his comrades taken from life, and I venture to say, caricatures of his first masters, among them naturally that of the teacher of drawing at the school, M. Pointurier; he was a jovial man not devoid of talent; he drew as

[February 28, 1923.]

naturally as he breathed, and allowed his pupils to develop faculties according to their natural bent. But Louis at the age of 13—the age of daring—dared to attempt a real portrait. Ah! if he could fix upon the paper the most familiar, the dearest, the loveliest of faces. . . . His heart tells him that though the busy grown-up folk will not find time to sit for him his mother will understand her child. The others have faces that are unknown and difficult, but the face of his mother is a landscape of which he knows every secret. "Oh, mamma, you must put on your pretty shawl and your Sunday bonnet!" Here is the portrait; nearly a century has passed over the powder of the pastel, but we see how seriously she played her part. She sits rather stiffly within the rigid folds of her plaid shawl; the perfect order of her coiffure is framed in an



House where Pasteur was born in Dôle (with a commemorative plate above the door).

impeccable bonnet. At a first glance her features seem rather severe, doubtless the length of the sitting had its effect . . . but if we look a little closer, do not her lips appear a little—very willingly—compressed? Can we not detect a desire to smile, a desire suppressed so as not to discourage her boy, who is struggling to do his best? I think so, for in the eyes glitters a happy light, which sees a little artist, silent, grave, absorbed in his work, dominated by the creative effort. . . .

And if we analyse this drawing, we see with what scrupulous care the child has brought out every detail, and here are the qualities which later on will guide the scientist.

Three years afterwards Pasteur, on the eve of leaving for the first time for

Paris, tried to catch the features of his father. As you will recognize, there is a certain poignant sadness in this portrait. Is it produced by the coming separation, or by a secret uneasiness due to the leaning towards an artistic career of a son for whom he dreamt of a more certain destiny?—for his ambition was to see his son a future rector of the college of Arbois! Or is it the bitterness of the ex-soldier of the Grande Armée who knows the vanity of glory? "I can still recall," said Pasteur forty years later, on looking at this portrait, "I can still recall this sadness of my father." This drawing is not signed. Doubtless, it remained unfinished. In my opinion it is the most impressive of Pasteur's drawings—the technique is more assured, and the analysis remains as moving in its keen, observant tenderness.

These two portraits call up the remembrance of some of the most touching words by Pasteur. In 1883 Pasteur, on visiting Dôle and seeing the house in



Pasteur's mother. (Pasteur del., aged 13.)

which he was born, and on which his fellow townsmen were fixing a commemorative tablet, invoked the memory of his father and mother, and all they had had done for him. Pasteur, deeply moved on remembering his early childhood, let his heart overflow, and to this emotion he gave expression in the following words:—

"O father, O mother, O my beloved ones who have gone before, you who lived so modestly in this little house, it is to you that I owe everything. Valiant mother, you have filled me with your own enthusiasm. And you, dear father, whose life was as hard as your work was hard, you have shown me what patience can do in continued effort. To look up, to learn beyond, to try to rise ever higher, that is what you have taught me. I bless you both, my dear parents, and yours be the homage done to this house to-day."

Shall we say that he owed his enthusiasm to his mother and his faculty of

hard work to his father? To his mother his gifts as an artist, and to his father his qualities as scientist? Let us beware of too subtle a discrimination. I have quoted these noble words of the son only to make you understand the deep meaning of these two pictures.

But to return to his career as an artist. He left home for Paris in 1836, but not for long—he could not bear to be so far from home—and his father took him back to Arbois, where his school work left him ample leisure for painting. He hesitates as to his vocation, a period of uncertainty in which he listens alternately to the voice of Art and to that of Science calling him at the same time. It is in this short time that he produced most of the drawings that you see here.

You see a vine owner with a young and refined face, wearing a stock like an ancient politician. And now the delightful portrait of a little boy, who in



Pasteur's father. (Pasteur del., aged 16.)

some measure recalls Velasquez. He is charming with his pink cheeks, and the likeness was such, that M. Vallery-Radot, who had never seen him, recognized, many years later, a distant cousin of the original in Arbois. Next the portrait of Mlle. Roch, No. 12, which might be called the "Lace Collar," in which we notice the same minute detail as in his mother's portrait. The very threads of the lace collar, the detail of the hair, are treated with the same minute care. You might think that the portrait is made up of several miniatures. The ophthalmologists here present have already diagnosed that these drawings are produced by short sight. I insist on this, because the eye for detail in the artist will be as sharp in the scientific man.

And here is the 80-year-old nun, Sœur Constance Parpandet, who was born in those times when George the Second was King of England. I do not care



so much for these more elaborate portraits. There is less simplicity and more craftsmanship about them.

We are now coming to the end of his career as an artist. He goes to the Lycée of Besançon in 1839. His drawing master takes a great interest in him and shows him *how* to draw, and at once he loses his personality. "I am making great strides in colour," writes Pasteur, "if not in resemblance." Oh the evils of teaching!

If a child is likely to develop too early, if he shows a disturbing originality, if he is threatened with genius, let him be sent to school. At school our artist loses his vigour. Look at the pretty—the too pretty—print of Chappuis, Pasteur's school-fellow. "I have never done anything so well drawn nor so like. The head master thinks it is very good!" (Yes, but it is the very opinion of the head master that disturbs me!)



Portrait of a nun, with the inscription, "*Sœur Constance Parpandet, âgée de 62 ans.*"  
(Pasteur del., aged 18.)

The last pastel is dated 1842. It was the portrait of Marcou, who afterwards taught geology at Cambridge, Massachusetts. Durant-Greville, who saw this portrait in America, wrote of it: "Many painters awarded medals at the Salon have never drawn or modelled a mouth with such precision. If Pasteur had wished he could have become someone of standing among painters, and—who knows?—perhaps a very great painter." In the year this portrait was drawn Pasteur entered the École Normale in Paris.

Is it not evident that Pasteur passed through a crisis? He loved his art passionately, but saw that his duty lay elsewhere. He broke off suddenly as if he had made a promise to himself to give up being an artist. If not, how are we to explain why he never re-opened his pastels box and why, he who had taken so much pleasure in drawing the children of other people, resisted



the pleasure of completing this collection of family portraits. That remains his own secret.

This does not mean that the artist was dead in Pasteur. Throughout his life he was faithful to his interest in painting; he never missed a Salon, and found time frequently to visit great national galleries. At Dresden, for example, his first visit—to the astonishment of his hosts, who awaited him at the laboratories—was devoted to the Museum. Among his intimate friends were many artists, among them the sculptors Paul Dubois and Perrault, the painters Henner and Eberfeldt. But Pasteur drew no more.

One of his friends who lived in daily intimacy with him, told me that he never suspected that the scientist was an artist, so much had scientific research monopolized his thoughts.

Without discussing the relations of Art and Science, I will only recall Pasteur's own expression and apply it to scientific imagination—"to learn beyond," and this sums up all Pasteur's work.

Pasteur is now at the École Normale, and here in the branch of Science in which he specializes his faculties as an artist are strengthened and developed. He remains a visionary, that is to say a mind in which the faculties of observation and imagination predominate. For instance, having taken up chemistry his curiosity bears on one of the most arid branches of this science and one in which investigation relies entirely on the eye, namely, optic chemistry: crystallography. His short-sighted scrutinizing eye, this eye of an artist as implacable as a sensitive film, will let nothing escape it, either the formation of the crystal, or its orientation, or the relation of its angles.

Where specialists, such as Mitcherlich, stop and fall back on the mystery of the racemates, a Pasteur *learns beyond* and discovers the mirror dissymmetry of the right and left tartaric acids. I believe that it is owing to his early analytical drawings that the artist has trained his eye to the analysis of crystals. In other words he has given us the portrait of his crystals with the same vigorous observation and passionate imagination.

"It was necessary to have seen Pasteur at his microscope," wrote Dr. Roux, "to form an idea of the patience with which he examined a preparation; moreover, he examined each object with the same minute care. Nothing escaped his myopic eye, and we used to say in jest, that he saw the microbes growing in the media." With the study of fermentations began the ascent: investigations of wine; silkworm disease; investigations of beer; virulent diseases; virus vaccines; the prophylaxis of rabies. Link by link the artist built, and the experimenter forged, the chain of genius.

At the celebration of the Centenary of Pasteur two months ago, at the Academy of Medicine, we were introduced to two different conceptions of Pasteur. President Béhal showed us a very human Pasteur, whose studies were not specially brilliant. "He was not a genius," said he. But Professor Widal looked upon Pasteur as one of those providential beings, created by nature to renew the orientation of science, and who was gifted with the spirit of intuition—a true genius. Of these two conceptions, the former is the more encouraging to us, and if he is the offspring of his work, of his method and of his will, there is no unbridgeable gulf between his mind and ours. "Pasteur soared above concrete experience. Without imagination the scientist would have stopped at experimental facts. Artist, he swept the infinite with a single cast of his line." That is what Professor William Bulloch has shown, when he speaks of "the work initiated with so much imagination and carried out with such incomparable skill by Louis Pasteur."

I trust that I have proved my point, and that I have succeeded in helping you to know Pasteur more intimately.

You know that Louis Pasteur rests in the crypt of the Institut Pasteur, a shrine worthy of him. On the door is one name only, but, above the tomb, the artist has engraved a quotation. The word "art" has the first place—deservedly. Here is the quotation: "Heureux celui qui porte dans son cœur un idéal de Beauté, et qui lui obeit, idéal de l'Art." "Happy is he who bears in his heart an ideal of Beauty and who follows it. The ideal of Art, the ideal of Science, the ideal of one's country, the ideal of the Virtues of the Gospel." These immortal words sum up the life of Pasteur, the Believer, the Patriot, the Scientist; and of Pasteur the Artist.

Sir HUMPHRY ROLLESTON: Your Excellency, ladies and gentlemen,—Your President has the duty of thanking those who have spoken to us to-night, but in one respect you will agree with me that he has not quite completed his duty, for he has said nothing about the charming address we had from him dealing with Pasteur as a man and as a medical pioneer. Among the many attractive points which your President made, none perhaps struck home more securely than that Pasteur owed much of his success to his wife, and the sympathy that existed between those two, thus showing that Pasteur the great man resembled some of us smaller men in a similar debt to their better halves. Now, not only do we owe a great deal to our President, to which he, of course, could not draw attention, but we owe a great deal to him and the Royal Society of Medicine for taking this opportunity of paying a compliment, so well deserved, to the genius of Pasteur and to the French. We are delighted that they have thus enabled us to-night to do what, with the most gracious courtesy, the Academy of Medicine of Paris did earlier this year in their celebration in honour of Edward Jenner. And it seems to me, and I am sure must seem to you, that this indeed should be a year of good augury for national friendship, for there have been these two celebrations: the centenary of Jenner's death, in Paris, and this celebration in honour of the greater Pasteur's birth a hundred years ago. Let us also express our admiration for the address of Professor Lowry, who has journeyed from Cambridge to give us this extremely clear account of the physico-chemical activities of Pasteur; and, perhaps most of all, to Dr. Monod, of Vichy, who has made the longest journey, for his brilliant speech on Pasteur, which had only one fault, its extreme shortness.

Sir STCLAIR THOMSON: It is a mere matter of form on my part to second this vote of thanks. You will be expected to show your appreciation by what the Bible calls "a cheerful noise"; but I feel quite sure that it is not by the noise you will make, but by the silence, the deep and profound silence which you have maintained during the last one and a half hours, that you demonstrate your appreciation of the speeches of this evening. They have shown to us Pasteur as a man, as a scientist, and as an artist. In his opening speech, the President said that science had no frontiers, and although we welcome science, whether it comes from the other side of the Rhine or from the other side of the Channel; and although we agree that science has no country, still scientific men have their nationalities, and it is a great delight to us to have had placed before us, in this exquisite address of your President, the personality of Pasteur, who is the greatest of Frenchmen. I was motoring through Savoy once with a French friend, Dr. Francois Helme, and his motor car broke down. While his chauffeur was putting it right, children gathered round the car, and Helme said it might interest me to see how French children were educated. He said to a little girl: "What standard are you in?" She replied, "The fourth." "That's all right. Now tell me who was the greatest Frenchman who ever lived?" What do you think she answered? "Louis Pasteur." Then he said, "Tell me the name of another great Frenchman, a great soldier," and she said, "Napoléon Premier.

Monsieur." Then he said, "Who was Shakespeare?" And she replied, "A great poet of England." That is a lesson for us to learn from France, their appreciation of great men; and it is a more striking lesson to know that their children are taught that the greatest of great Frenchmen was Louis Pasteur.

(The resolution was carried by acclamation).

Sir WILLIAM HALE-WHITE (President): I speak on behalf of the distinguished colleagues who have addressed you when I say that we all three thank you most heartily for having passed this vote of thanks.

## The Royal Society of Medicine.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### OCCASIONAL LECTURE.

#### The Urgent Need for Education in the Control of Cancer.

By JOSEPH E. ADAMS, M.S., F.R.C.S.

I OUGHT, perhaps, at the outset to offer a word of explanation as to how I came to find myself in this position. In September of last year I wrote a letter to the *Lancet*, under the heading "Cancer and the Public," in which I suggested that the time had come when a more determined effort ought to be made to let the public, that is the potential sufferers, know what is known about cancer and how it may be recognized; that although no one knows its cause, there are ways of curing cancer. I further suggested that a Society for the Control of Cancer, similar to those in existence for fighting tuberculosis and venereal disease, ought to be established. The title of my letter was borrowed from the lay press; the society would be a copy of one which already exists, and is very active, in America. I lay no claim to originality, but I trust I can put before you an idea as to how this desirable work of propaganda can be begun.

But first it is essential to know that the medical profession is of opinion that it should be done, therefore I thank you in advance for coming to this meeting so that a clear view may be expressed.

One of the characteristics of our profession is that whilst it has a body and a truly great soul, it possesses no actual head. It supports a number of societies and associations each with somewhat different functions. I hope I shall not give offence to anyone, and I am sure I shall not offend my audience, if I say that the Royal Society of Medicine represents the intelligence of the medical profession. Let us hope that what Lancashire is said to be to political England, No. 1, Wimpole Street is to the practitioners of medicine. Nothing that I suggest ought to be done unless, and until, the medical profession as a whole approves of it. I do not anticipate active opposition, but there is a worse enemy, and that is apathy.

#### THE NEED FOR EDUCATION.

That cancer is on the increase, that it has ceased to confine its attentions to the middle-aged or old, that it accounts for one in every thousand deaths recorded in England and Wales by the Registrar-General, are facts well known to you. Another melancholy fact is that we know very little more about its

causation in man than we did twenty years ago. Does all this mean that we are powerless against man's strongest pathological enemy? Does it not rather suggest that we must try new methods? We know that the majority of patients whom we encounter either in private or public practice come to us too late for a reasonable chance of cure. Sometimes we are moved to ask the patient why he or she did not come before. This is really an ingenious dodge for putting the blame on to innocent shoulders. We have not encouraged patients to come sooner. We have not proved that cancer is a curable disease. We have not taught them what are the danger signals. And our chief excuse is that we do not know enough about it. Yet we do know of many conditions that predispose to cancer, and, without treading on controversial ground, I think I may mention syphilis, chronic degenerative mastitis, and unhealed lacerations of the uterine cervix. Are these conditions treated as energetically as their relationship to cancer demands?

#### WHOM SHOULD WE INSTRUCT?

Of women, as you know, cancer takes a heavier toll than of men. Their generative organs are peculiarly liable to attack. In a sort of subconscious way they know this and they appear to accept it as the curse of Eve. Bravely indeed do they submit to mutilating operations, but surely these could be minimized if only every woman aged over 40 knew that a lump in her breast is ten times more likely to be malignant than simple. In my experience a woman with a lump in her breast thinks it means nothing unless it is painful. Conversely, a woman with a painful breast makes light of it until she finds a lump. This accounts for the fact that the average duration of history, at any rate, amongst hospital patients, in cases of carcinoma of breast is at least six months. What appalling ignorance this reveals! What a terrible handicap to successful treatment!

Middle-aged men are apt to think that indigestion and piles are the inevitable result of a sedentary life, and that a holiday, a bottle of medicine, or a few pills will certainly put them right. Have not some of us been guilty of aiding and abetting this error by treating patients without a thorough examination? Has not the comfortable diagnosis of piles been made too often where a rectal examination would have proved the presence of a growth in the bowel as the source of bleeding, or the primary cause of symptomatic piles? Surely by this time it ought to be recognized by the public that treatment without examination is not worth paying for, and by the medical profession that it is not worth carrying out. Of course if investigation is declined by the patient he or she alone is responsible.

I might multiply these examples, but to me it is clear that the public need enlightenment and the medical profession encouragement. Both should recognize that diagnosis must precede treatment.

It is far from my intention to decry the work of scientific research, and I think one is justified in assuming that the discovery of the cause of cancer will be made. How, and under what circumstances, no man can at present tell. Much knowledge has already been accumulated by our Imperial Cancer Research Fund and I do not believe that a penny of their money has been wasted, or a minute of their time. In experimental cancer undoubtedly considerable advance in knowledge has been made within the last few months. But because we are unable as yet to tackle cancer on the large scale is it any reason why we should not attempt to do so in a small way?

## HOW CAN CANCER EDUCATION BE CONDUCTED?

In order to consider this aspect of the question it is necessary to give some details of the American Society for the Control of Cancer, and to judge how far their methods are appropriate in Great Britain. This society was founded in New York on May 22, 1913, at a meeting of delegates appointed by the principal medical societies of the United States, together with a number of prominent citizens, who were convinced of the need of a national organization "to disseminate knowledge concerning the symptoms, diagnosis, treatment and prevention of cancer, to investigate the conditions under which cancer is found, and to compile statistics in regard thereto." The movers in the matter were the American Gynæcological Society. Active work began in January of 1914. The personnel of the Society includes names which are well known and respected on both sides of the Atlantic: William Keen, Joseph Bloodgood, William Mayo; also George Armstrong, of Montreal, just to mention a few. The movement is not therefore limited to the United States.

During the war the work of the society was damped down, but after 1918 Dr. Charles A. Powers,<sup>1</sup> a well-known surgeon of Denver, Colorado, was appointed President and he has made things move with rapidity. Having retired from active practice he devotes himself entirely to this work. Every State now has its organization with a leading doctor as chairman, and the branches act as liaison agencies between scientific investigators of cancer problems and local social communities; they are popularizers of science in this respect. They issue pamphlets concerning cancer clothed in language that the man in the street can understand, and they distribute these leaflets broadcast by means of committees and health workers. Lectures are also given, and one of their proudest achievements was a "National Cancer Week" conducted throughout the United States from October 30 to November 5, in 1921. Six hundred thousand persons were then lectured to and several thousand were reached by short addresses in churches and theatres. No less than five million pieces of literature were distributed and the newspapers and magazines were pressed into service. Advertisements by posters, and films in picture palaces, were also employed; in fact, no means of reaching the public ear appears to have been neglected. At least ten million persons are supposed to have received information of the vital facts of cancer control during this short period of seven days. A second "Cancer Week" was held in November of last year. This work is carried on in co-operation with State and City Health Departments and the American Red Cross Society. Literature on the subject is gladly posted to any person on request. All this costs money and apparently the sinews of war are obtained entirely from voluntary donations and subscriptions.

America has been called the "land of the dollar," but is it not also the "land of brains"? We have borrowed the dollars and are now struggling to repay the loan. We have a call on the brains without the same obligation. Nothing would delight the Americans more than that we should imitate them, and I imagine that they are very susceptible to this form of flattery. In this country it is a good and valid reason for the performance of a surgical operation to say that that operation is done in Rochester, Minnesota. Would it not be a good reason for cancer education to say that it is done all over America?

<sup>1</sup> Since this was written I have heard from America that Dr. Powers died in December of last year.

## CANCEROPHOBIA.

So far I have said nothing about one obvious objection to wide dissemination of knowledge of cancer. This objection is that if people's minds are so agitated there will be many who imagine that they have cancer without any justification; that their state may be worse than that of the sufferers from the disease itself. As a proof of this I am told that syphilophobia is a truly direful disease. It may be, but is there not a moral reason for this which would be entirely absent with regard to cancer? I have the authority of Dr. Powers, the President of the American Society, for stating that cancerophobia is not a serious trouble, and that in his own city of Denver, Colorado, which has been thoroughly instructed, cancerophobia hardly exists.

## WHAT HAS BEEN DONE IN THIS COUNTRY.

Even in this country some work has been done along the lines I am suggesting, and seventeen years ago a book was published by Mr. Childe entitled "The Control of a Scourge. How Cancer can be Cured." As he is to open the discussion, I must not anticipate, but he tells me that the publishers are asking for a second edition, so there is one indication that the time is opportune for cancer education. Medical officers of health in various parts of the country are endeavouring to inform the public, and pamphlets are being issued from various hospitals. These efforts are most praiseworthy, but they can only succeed in reaching a very small proportion of those who are exposed to the danger of malignant disease. Remember that one in every thousand dies of the disease annually in England and Wales; the incidence must be far greater than the mortality.

It must have been apparent to all of you that the lay Press is also anxious to inform its readers on health topics. Sometimes it does so in a discreet and useful fashion, but its efforts require scientific direction.

## WHAT CAN BE DONE.

My original suggestion was that we ought to have a Society for the Control of Cancer, similar to that working, by every conceivable means, in America. I recognize, however, that we are a poor country at the present time, and such a society may be a luxury which we can ill afford. Fortunately there is machinery at hand which can be utilized for the purpose. We have with us this afternoon the heads of the British Red Cross Society. I refer to Sir Arthur Stanley and Sir Napier Burnett. Now these two gentlemen have kindly come here this afternoon, and, convinced as they are of the potential value of the work, they are prepared to help in every possible way, provided the doctors want the work done. One of the charms of the British Red Cross Society is that it has money, and I can conceive of no better peace time occupation than that of helping to fight cancer. This society has an organization by which it can reach a large portion of the public by lecture, by instruction in classes, and by pamphlet. To include the dissemination of knowledge of cancer and its early signs and dangers would not be difficult. They have people who could translate our heavy scientific verbiage into commonplace everyday language, and they are ready to do so, and to broadcast the result.



All that is really needed to set this useful machinery in motion is willingness on the part of the medical profession, and a specific body of them who are prepared to act as a standing committee pledged to supply facts with regard to cancer which will help to educate, when put in a form which the public can grasp.

As a practical outcome of this meeting I hope such a committee, and it should be a small committee of real live people, will be formed. It is essential that it should act impersonally, and I think the choice of the personnel might well be left to the Council of the Royal Society of Medicine. One of the things to be avoided is any appearance of even "oblique advertisement." I regard this new phrase as a rather unfortunate invention, but we must be circumspect, and the pact between the committee and the Red Cross Society should be that nothing be published except with the imprimatur of the committee, and that all names should be withheld except that of the Royal Society of Medicine.

#### MINISTRY OF HEALTH AND INSURANCE SOCIETIES.

There are just two other points I should like to touch upon. The first is that one might naturally suppose that the Ministry of Health might help in such a good cause. I imagine that it would, at least by its blessing, if only we can establish the goodness of the cause. Secondly, there are financial interests that would benefit by anything done to lessen the incidence of cancer. I refer to the life assurance companies. Surely, if it pays the fire insurance companies to run a salvage corps, it should be worth while for life assurance societies to assist in the salvage of human lives, and these companies might see that it is to their advantage that such public education should be carried on. The start, however, must be made by those who know the dangers of delay, and who believe in the value of instruction. Above all, let us not wait until the Registrar-General's figures reach a total of fifty thousand deaths per annum.

With your permission, Mr. President, I should like in due course to propose the following resolutions:—

#### PROPOSED RESOLUTIONS.

- (1) That it is desirable that the public should be given more information as to the early signs of cancer and the prospects of cure by immediate treatment.
- (2) That the British Red Cross Society should be asked to conduct this publicity campaign by means of lectures and pamphlets.
- (3) That the Council of the Royal Society of Medicine be requested by this meeting to nominate a Standing Committee to supply information to the British Red Cross Society suitable for wide dissemination and the education of the public.

Dr. LAPTHORN SMITH seconded the adoption of the resolutions which were duly carried.

## DISCUSSION.

Mr. C. P. CHILDE said that it was particularly gratifying to him to be present at the meeting, because it was proposed to do that day exactly what he advocated seventeen years ago in a book he wrote for the New Library of Medicine. This had been published both in this country and in America and he had had, as a result of it, many communications with various people in America on the subject. He had often thought it might have been the germ of the Society for the Control of Cancer founded six years later in the United States. It was significant of the changed attitude of the public towards the discussion of disease in general and cancer in particular, that the publishers at that time had been afraid to put the word "cancer" on the cover of the book, but that now, when they were asking him (Mr. Childe) to bring out a second edition, they had no objection to some such title as "Cancer and the Public."

The only things known about cancer which were worth knowing were its age-incidence, its association in some way with chronic irritation, and the fact that it was in the first instance a local disease, and therefore capable of cure if removed early enough—and they were meeting that night to consider whether anything could be done, and if so what could be done, to secure that people suffering from cancer in some accessible regions should be treated earlier than was the case at the present time. That brought him to the subject of education, more especially the education of the medical student. He spoke without exact knowledge as to present teaching at the schools, but in his opinion if a student up for his diploma were asked what he would do with a tumour in the breast of a woman over 35 or 40 years of age, and gave any other answer than that he would remove the piece of breast containing the tumour and have it submitted to microscopic examination, unless he was absolutely certain it was *not* cancer, that student should be sent down for six months. Or again, if asked what he would do in the case of a woman about the same age with irregular hæmorrhage, or any abnormal hæmorrhage, and he gave any answer other than that he would put her under an anæsthetic, dilate the cervix and exclude cancer, unless an ordinary examination revealed the fact that she was certainly *not* suffering from that disease, he should be rejected until he knew better. Other similar instances would occur to those present. The teaching should be—especially in patients after middle life—to exclude or confirm cancer at once, without any delay and before any treatment. This teaching should be absolute and without any exception whatever; and unless the student had unequivocally mastered it, he ought not to be granted a licence to practise. The next consideration was the education of the public, the potential victims of cancer, and this was the immediate object of the meeting. It was known that in some regions of the body cancer was curable. Every operating surgeon could produce cases of cancer, proved to have been that disease, in which the patients, after operation, had lived to the ordinary span of life and never had any return of the disease. He (Mr. Childe) was no believer in the three or five years' limit, or in any limit, because apparently the disease might return after almost any lapse of time, and no one was therefore ever justified in promising a cure. But the results to which he had just alluded were a good enough cure for the patient and were all that he wanted. Yet although the disease was curable in many instances, as a matter of fact very few were cured, and the reason so few were cured was that patients hardly ever applied to have the disease removed early enough to give them even a reasonable chance of cure. He could quote statistics to prove this and they were given in his book *in extenso*. The question was whether the public could be given any information which would enable them to apply in time. The only objection was the possibility of aggravating "cancerophobia." Personally he did not attach much weight to this. Cancerophobia existed at the present time; he supposed there was nobody present who had not been consulted by people convinced that they had cancer, and who had not a sign or symptom of it; and the reason was that the public believed it to be such a fatal disease that there were some nervous people who if they had an ache or pain anywhere immediately thought it was a sign of cancer. As a matter of fact an ache or a pain was never

a symptom of early cancer at all—but if the public could be brought to believe that if they applied in time cancer in many regions of the body was not such a fatal disease after all, and that they could be cured, was it likely that cancerophobia would be increased? On the contrary, it would surely be lessened, and the public would have less dread of it than they now had. Of course, if it was decided to give the public information, they must just be taught some very simple facts about cancer in its common and obvious situations. It was no use endeavouring to aim at an elaborate education on cancer of the intestines and other internal organs, or cancer occurring at exceptional ages, &c.: this would be worse than useless. What should be made generally known were such simple straightforward facts as the following: Every woman should know that if she found a lump in her breast and she was over 40 years of age it was almost certainly cancer, however well she felt and although she had no pain whatever; and that if she applied at once, and did not watch it for six months, it was curable and her life could be saved. Similarly every woman should know that if she had passed the menopause, and saw a little blood staining her clothes, however well she felt and although she had no pain whatever, she had in all probability cancer, and that if she applied at once she could be cured. It was no use being elaborate. The information should consist of perfectly simple facts with regard to cancer in obvious situations. Fortunately four out of five of the most common situations of cancer were obvious situations—i.e., they gave danger signals which must be apparent to the patient, namely, the lip and tongue in men and the breast and uterus in women, and it was on these that the information given should be concentrated.

Lastly, as to the method of disseminating information. There were various ways and much could be done that was not done now, even without actually giving direct information to the potential sufferers from cancer if that was considered going too far. For instance all nurses and all midwives should have this information at their finger tips and it should form part of their training and curriculum. Nurses and midwives were frequently consulted by women as to a lump in the breast or irregular bleeding, and should be in a position to clinch the matter at once, and send their questioner without any delay to a doctor. Also the St. John Ambulance Association might give lectures, similar to those given on simple emergencies, on the early signs of cancer in obvious situations. The people who should attend these lectures were clergymen, clergymen's wives, health visitors, district nurses and others who were constantly brought into touch with the poor and ignorant. Other channels of information would no doubt be devised if such a campaign were put in hand. He (Mr. Childe) need hardly say that it would be peculiarly gratifying to him if, seventeen years after drawing attention to this matter and advocating a very similar campaign to that now suggested, he saw such a project put in hand.

LORD DAWSON OF PENN said that in focussing this all-important problem the fact should never be lost sight of that research into the causes of cancer must always stand in the forefront of the programme. But while the results of that research were awaited, some means must be found of getting existing cases under treatment as early as possible. It was necessary to bring home to the profession, in the first place, the need for a closer appreciation of the early symptoms of the disease, and he believed that, in fact, considerable attention was paid to this matter in the medical schools at the present time. When, however, the qualified man went out to take up his practice he was deprived alike of the stimulating atmosphere and of the equipment to which he had been accustomed in the hospital. Often he had not at his command those ancillary means of diagnosis which were of such great value, and the effect of this deprivation was not only to limit his powers, but slowly to discourage his zeal. Until hospital and clinical equipment was made accessible to practitioners in all parts of the country the movement to control cancer would be handicapped. If the medical men were educated up to the great importance of this subject and were given opportunities and encouragement, it would have a repercussion upon the public itself (because good doctors always meant sensible patients), and would go a long way towards meeting the end in view.

So far as the direct instruction of the public itself was concerned, there were two

lines of action to be considered. One was a direct and specialized propaganda against cancer. As to the value of this he had some doubts. The more useful plan had been suggested by Mr. Childe. It was to educate the public generally in health matters, by means of lectures on health in which variations from health and the signs of such variations would be discussed, but in which the special application of a particular symptom to a particular disease would be avoided. This last was the function of the doctor in dealing with his patient, not that of the health lecturer addressing the public. The significance of the variations should be left to the doctor, whom the people concerned should be encouraged to consult. He saw a real danger in cancerophobia, but by the methods just indicated he thought it could be avoided. He was distrustful of intensive methods of propaganda; they might be successful from one point of view, and yet deleterious in other respects. It was not enough to be intensive and energetic, it was equally necessary to be wise. The means by which it was proposed to educate the public should be selected most carefully. The best means would be by lectures. If broadcasting and cinemas were made the vehicle—possibly even sky-signs also—the effect would be to create an unhealthy atmosphere, and to concentrate public attention on lurid and morbid subjects so that more harm than good might be done. The anti-venereal campaign should not be too closely copied. That campaign was started under very special circumstances which did not apply in the present instance, and he thought that even in the case of the anti-venereal campaign the time had come to transfer propaganda from the streets to the lecture hall, where this other propaganda should begin.

Dr. HERBERT SPENCER said that sixteen years ago he read a paper at the annual meeting of the British Medical Association at Exeter, dealing with measures to be recommended to secure the earlier recognition of uterine cancer. The views expressed in that paper were received with a unanimity rare in professional discussions. One line of action which he advocated was to bring forward for demonstration to the students in the medical schools patients who had remained for twenty or twenty-five years free from recurrence. This would effectively counteract the pessimism which existed in the public and to some extent in the professional mind with regard to the possibility of curing cancer. Even the smallest town ought also to have its cottage hospital where visiting consultants would be available to examine patients referred for suspected cancer. In the paper referred to he had mentioned means of educating the public and thought that could be best done by means of leaflets issued by medical officers of health, health visitors, midwives and nurses. He did not favour widespread propaganda by means of the Press. He thought it was most important to educate students in the means of diagnosing cancer, and for that purpose the number of gynaecological beds in every hospital with a medical school should be at least fifty. In the mortality of cancer of the uterus some improvement could be seen, as shown by the Registrar-General's Report for 1900, 1915, and 1920 respectively.

## REGISTRAR-GENERAL'S REPORT.

		Female population		Cancer of uterus		Cancer of ovary		Cancer of breast
For year 1920	...	19,658,000	...	4,090	...	603	...	4,488
" 1915	...	19,365,342	...	3,899	...	562	...	3,920
" 1900	...	16,663,706	...	3,679	...	275	...	2,570

from which it was seen that mortality from cancer of the uterus had not kept pace with the increase in population. On the other hand, deaths from cancer of the ovary had enormously increased and cancer of the breast had displaced cancer of the uterus as the most frequent site of fatal cancer in women.

The Honourable Sir ARTHUR STANLEY said that if the British Red Cross Society was asked to undertake the educational work under discussion it would be glad to do it, and it had the necessary machinery for carrying out such work.

Sir NAPIER BURNETT said that the Red Cross Society would readily agree to carry out any such work in this connexion as the Royal Society of Medicine might request it to do.

Mr. CECIL ROWNTREE said that Mr. Adams' stimulating and thoughtful address had provoked the complete unanimity that might be expected, regard being had to the fact that they were all alive to the seriousness and urgency of the cancer problem and to the importance of educating the public and the profession as to the value of adequate surgical treatment in the early stages of the disease. But he (Mr. Rowntree) could not help feeling that Mr. Adams and other speakers had been looking at the question somewhat too exclusively through the surgical end of the telescope. It was the end he usually affected himself, and he heartily agreed that in many kinds of cancer early diagnosis meant everything between life and death; but of what avail was it to educate the public to the pitch of recognizing the early stages of cancer of the œsophagus, liver, lung or pancreas, or the first signs of melanoma or periosteal sarcoma? Could anyone present save the lives of patients in these cases be they ever so early?

No, the real cancer problem was the problem of its cause and cure, and no amount of educational propaganda or legislative enactments would help in this direction; they had got to help themselves. Lord Dawson had referred to the importance of cancer research, and he (Mr. Rowntree) believed that what was wanted was, firstly, more generously endowed, and, secondly, more intensive and more highly organized research work, for it really seemed as if cancer research was suffering from too many watertight compartments, and that there was far too much overlapping. Some laboratories had more clinical and pathological material than they could handle, while others were quite divorced from clinical material and experience. Some were poor and struggling, others were rich and struggling for more, but the net result was duplication of negative observations and the holding up of important investigations for the laborious working out of some of those minor side-issues that so constantly cropped up in all research work, but which had perhaps already been completely solved by some other laboratory round the corner.

It might be said that this was all mere destructive criticism, and some of it perhaps untrue. The alternative to the present state of affairs appeared to be the organization of some central body which would to some extent guide, advise upon and control research, discipline researchers, ration material, and in fact, exercise just such a general superintendence and influence as was at present done in other spheres of research by the Medical Research Council. The power of such a body would have, of course, to be rendered real and actual by the possession of a central fund with which, by means of grants in aid or in reward, it could initiate special lines of research and recompense achievement. He did not, of course, suggest for a moment that the central body should exercise any direct financial or other control over any individual laboratory, nor was he suggesting any pooling of staffs or merging of the identity of competing institutions, for in that way lay bureaucracy and the death of originality and effort. But there would naturally have to be some sort of responsibility, some sort of periodical report of the kind, quality and quantity of the work proceeding in each laboratory, and some method of pooling and publishing the progress of experiments. But the autonomy of each and every laboratory that pulled its weight must be maintained, and with it that independence of thought and judgment which was so important a characteristic of the true researcher.

Dr. S. MONCKTON COPEMAN, F.R.S. (Ministry of Health), said that his Ministry had lately appointed a Departmental Committee on Cancer, of which Sir George Newman was Chairman, and on which, in addition to the official members, were representatives of various bodies who were interested in the subject from the clinical and pathological aspect. This Committee would necessarily have to consider the question of public education in the control of cancer. He suggested that perhaps the Royal Society of Medicine might think it desirable to consult with the Government Committee before taking any special course of its own. In the event of it being

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decided that the education of the public should be put in the forefront of any programme directed to the control of cancer the public health services of the State (including nurses and health visitors) should all be utilized for this purpose.

Dr. A. MEARNS FRASER (Medical Officer of Health, Portsmouth) said that to those who, like himself, were engaged in public health work, the subject was of particular interest from the fact that cancer was gradually occupying the most prominent position in their death returns; for instance, during the period of twenty-five years odd that he had been Medical Officer of Health for Portsmouth, the proportion of deaths from cancer to the total number of deaths registered had increased from 1 in 20 to 1 in 10.

Unfortunately, in spite of extensive research, both the cause and the method of the spread of cancer were still unknown, and no one was therefore in a position to issue any advice in regard to the prevention of this disease such as could be given in regard to small-pox, typhoid fever, scarlet fever, and other diseases. But although it was not possible to take any action in regard to the prevention of the *incidence* of cancer, it might be possible at least to secure a reduction of the *mortality* from it, and it might interest this meeting to know the steps in this direction which had been taken by the Portsmouth Town Council, who, it might be added, in this matter acted on the initiation of Mr. C. P. Childe, who had for some time been the Chairman of the Health Committee.

The action taken had been based upon the assumption that the only known measures which at present offered any hope of curing cancer depended on the early total removal of the cancer, before it had had time to infiltrate surrounding tissue and glands to an extent that rendered complete removal impossible. They had been assured by surgeons that the principal cause why operative treatment so often failed was because patients delayed consulting a medical man until it was too late for surgery to have a reasonable hope of success. They had been informed that it was of paramount importance to make the public appreciate first, that certain symptoms, which in themselves often caused no alarm and were regarded as trivial, were really the first indication of the presence of cancer; and, secondly, that at this early stage and in many situations in the body, the disease lent itself to successful surgical interference.

To this end, therefore, leaflets, of which he (Dr. Fraser) showed a specimen (*see* Appendix), had been prepared and issued to the public by the Health Department and also distributed through certain special agencies such as health visitors, midwives, social workers, &c. In addition, every month the leaflet had been published in the advertisement columns of the local Press, and addresses on the subject had been given to health visitors, nurses, visitors, and others to whom it was thought the information would prove useful. The leaflet very briefly called attention to various suspicious symptoms which, appearing in middle life, indicated the possibility of the presence of cancer, and urgently warned all persons in whom such conditions might appear not to delay in consulting a medical man. The one object aimed at had been to educate the public to get as early treatment as possible, and not to allow suspicious conditions to continue unheeded without getting proper medical advice as to their significance.

The above measures had been decided upon by the Portsmouth Town Council in 1913. Unfortunately, owing to the general disturbance caused by the war, they fell into abeyance but at the time there was ample evidence from medical men practising in the town that members of the public were being reached and the patients in many early cases of cancer did undoubtedly consult doctors because of the propaganda work of the Health Committee.

Lord Dawson in his interesting remarks had rather spoken against propaganda work and had urged that more harm than good was likely to be caused by lurid publicity campaigns, including cinema shows and other methods, such as had been organized in connexion with tuberculosis and venereal disease. He (Dr. Fraser) agreed that a number of the methods which had been adopted, especially in regard to venereal disease, were objectionable and in no way serviceable; still, it must be remembered that there was a medium in all things, and he believed that there was a possibility of considerable saving of life by means of propaganda work which might be made effective without infringing the canons of good taste. It was the



hope of all that the exhaustive research work now being carried out might eventually result in the discovery of the cause of cancer, but in the meantime the toll from this disease was so appalling that they could not afford to sit still and do nothing, and totally neglect any suitable educative measures which offered reasonable grounds for securing a reduction in its mortality.

He (Dr. Fraser) suggested that the first practical step for the control of cancer was an authoritative pronouncement on the subject to be made by such a representative body of medical men as would carry weight both with the profession and with the public. Such a statement might possibly endorse such measures as had been adopted at Portsmouth and other places—on the other hand it might make totally different suggestions. But, whatever the statement, it was to be assumed that it would embody the best available measures which could be suggested for securing a reduction in the present cancer death-rate. Once this authoritative pronouncement had been made the next step would be the adoption of measures to bring its recommendations to the notice of the public, i.e., propaganda work. It had been suggested that the Red Cross Society and the St. John Ambulance were suitable bodies for this purpose; doubtless this suggestion had much to commend it; but surely the essential bodies above all to enlist in any campaign were the local health authorities; these were already possessed of medical and sanitary staffs and had all the machinery for getting in touch with the public ready to hand. Moreover, as evidenced by several isolated instances, many of these bodies were already convinced of the urgent necessity for action and were taking steps in one direction or another to try to secure some control over cancer. He suggested to the meeting, therefore, that the most effective propaganda work for the control of cancer could be secured only through local health authorities. He also suggested that if some such authoritative statement as he had indicated could be issued by the medical profession he felt certain that the Ministry of Health would take the necessary steps to see that it was brought to the notice of the local sanitary authorities, who at the present time were only too anxious to do something to reduce the awful mortality from this dread disease.

#### APPENDIX.

*[Copy of Leaflet issued by the Health Department, Portsmouth.]*

#### BOROUGH OF PORTSMOUTH.

#### CANCER.

#### SPECIAL NOTICE TO THE PUBLIC.

THE following leaflet is issued by the Portsmouth Health Committee, because so many persons die from cancer whose lives could be saved if they acted upon the advice here offered. The importance of this subject to the public is shown by the fact that of all persons over 45 years of age, one in ten dies from cancer. Issued by the Health Department, Portsmouth.

#### CANCER.

*It is vitally important that the following facts about Cancer should be known.*

It has been brought to the notice of the Health Committee that of the number of persons who die each year from cancer many could have been cured if they had applied earlier for medical advice. On questioning patients as to why they did not apply to a doctor earlier, the reason almost invariably given is that as the early symptoms were unaccompanied by pain, it was not thought that anything serious was the matter.

In order, therefore, to call the attention of the public to the significance of certain symptoms and conditions, and to the vital importance of acting promptly on the occurrence of these, it has been decided to make the following facts public.

The only cure for cancer, at present known, is its early and complete removal. Cancer, if removed early, has been proved conclusively to be a curable disease. If neglected, and not removed in its earliest stages, it is practically invariably fatal. The paramount importance of its early recognition and early removal is therefore evident. For this purpose the assistance both of the public and the medical profession is requisite; and a grave responsibility rests on both. It is only by their mutual co-operation that the ravages of this terrible disease can be lessened. The following information should be of vital assistance to the public. It is no exaggeration to say



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that, if acted upon, the result would be the saving annually of many hundreds of lives, which at present are inevitably lost.

(1) Cancer, in its early and curable stage, gives rise to no pain or symptom of ill-health whatever.

(2) Nevertheless, in its commonest situations, the signs of it in its early stage are conspicuously manifest. To witness:

(3) In case of any swelling occurring in the breast of a woman after 40 years of age, a medical man should at once be consulted. A large proportion of such swellings are cancer.

(4) Any bleeding, however trivial, occurring *after* the change of life means almost invariably cancer, and cancer which is then curable. If neglected till pain occurs, it means cancer which is almost always incurable.

(5) Any irregular bleeding occurring at the change of life should invariably be submitted to a doctor's investigation. It is not the natural method of the onset of the change of life, and in a large number of cases means commencing cancer.

(6) Any wart or sore occurring spontaneously on the lower lip in a man over 45 years of age is almost certainly cancer. If removed at once the cure is certain, if neglected the result is inevitably fatal.

(7) Any sore or swelling occurring on the tongue or inside of the mouth in a man after 45 years of age should be submitted to investigation without a moment's delay, and the decision at once arrived at by an expert microscopical examination as to whether it is cancer or not. A very large proportion of such sores or swellings occurring at this time of life are cancer, and if neglected for only a few weeks the result is almost inevitably fatal. If removed at once the prospect of cure is good.

(8) Any bleeding occurring from the bowels after 45 years of age, commonly supposed by the public to be "piles," should be submitted to investigation at once. A large proportion of such cases are cancer, which at this stage is perfectly curable.

(9) When warts, moles, or other growths on the skin are exposed to constant irritation they should be immediately removed. A large number of them, if neglected, terminate in cancer.

(10) Avoid irritation of the tongue and cheeks by broken jagged teeth, and of the lower lip by clay pipes. Many of these irritations, if neglected, terminate in cancer.

(11) Although there is no evidence that cancer is communicable under ordinary circumstances it is desirable that rooms occupied by a person suffering from cancer should be cleaned and disinfected from time to time.

Health Department, Town Hall, Portsmouth.

January, 1914.

A. MEARNS FRASER, M.D.,

Medical Officer of Health.

*Further copies of this notice may be obtained on application.*

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### CORRIGENDUM.

The following note was inadvertently omitted at the end of the Pasteur Centenary Report (see *Proceedings*, No. 6, April, 1923):—

"For the loan of the blocks illustrating the account of the Pasteur Centenary the Society is indebted to the courtesy of the *British Medical Journal*."

PROCEEDINGS  
OF THE  
ROYAL SOCIETY OF MEDICINE

EDITED BY  
SIR JOHN Y. W. MACALISTER  
UNDER THE DIRECTION OF  
THE EDITORIAL COMMITTEE

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**VOLUME THE SIXTEENTH**

SESSION 1922-23

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SECTION OF ANÆSTHETICS



LONDON  
LONGMANS, GREEN & CO., PATERNOSTER ROW  
1923

## Section of Anæsthetics.

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DR. J. H. CHALDECOTT (p. 33), Mr. I. HAMILTON BEATTIE (p. 38), Dr. MENNELL (p. 39), Mr. H. R. OSWALD (p. 39), Mr. FINUCANE (p. 40), Dr. H. P. CRAMPTON (p. 40), Mr. BOYLE (p. 41), Mr. MORTIMER (p. 41).
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## Section of Anæsthetics.

President—Mr. A. L. FLEMMING, M.B.

### The Anæsthetization of Patients for the Classical Cæsarean Section.

By HERBERT R. SPENCER, M.D., B.S., F.R.C.P.

CÆSAREAN section has a special interest for anæsthetists and obstetricians from the fact that the welfare and life of two or more individuals are involved.

On the obstetrical indications for the operation I will say nothing on this occasion except that they are being extended too widely by many practitioners.

On the anæsthetic aspect it requires some courage for an obstetrician to speak before an audience of specialists in anæsthetics, and my courage might have failed me had I not been convinced by an experience of thirty-five years of the value and harmlessness to mother and child of small quantities of chloroform given *à la reine* to mitigate the pains of labour, while insisting on the necessity of anæsthetization by an expert anæsthetist whenever an operation is required.

Perhaps I may claim a little indulgence for having broken a lance in behalf of your speciality at the Centenary of Ovariectomy meeting of the American Gynæcological Society at New York in 1909, on which occasion, in a discussion on anæsthesia,<sup>1</sup> I expressed my amazement at finding that American patients were anæsthetized by nurses instead of specially qualified medical practitioners. My criticism was well received by my American colleagues; but Dr. Clarence Webster, of Chicago (formerly of Edinburgh) was so affected by my accurate description of the position of anæsthetists in London that he informed the meeting that Scotland was the most important part of the United Kingdom, that in 1896 there was only one paid anæsthetist in Edinburgh and that patients were generally anæsthetized there by medical students.

In this communication it is to be understood that the anæsthetic is to be administered by specially trained qualified medical practitioners.

Of the two chief methods of anæsthetizing a patient the inhalation methods are generally preferable in cases of Cæsarean section.

Spinal anæsthesia is undoubtedly more dangerous than inhalation anæsthesia and has drawbacks from which inhalation anæsthesia is free.

Infiltration anæsthesia has advantages, particularly in the case of patients with pulmonary or cardiac affections. I used it successfully twenty-two years ago<sup>2</sup> in Cæsarean section repeated for the third time. But I have not employed it since. Spinal anæsthesia and infiltration anæsthesia are less humane than inhalation methods, which induce complete loss of consciousness; and, although they are useful in certain conditions, they are not advisable as a routine method, which it is the object of this short communication to advocate.

Of the two chief inhalation anæsthetics, chloroform and ether, I suppose no anæsthetist will assert that chloroform is as safe as ether, when administered to the surgical degree for long periods, especially to pregnant women. It has therefore largely been given up in favour of ether administered either by the "open" or "closed" method.

*The great disadvantage of ether is that it leads to asphyxia of the infant, which is not the case with chloroform administered to the minimal surgical*

<sup>1</sup> *Trans. Amer. Gyn. Soc.*, 1909, p. 304.

<sup>2</sup> *Journ. of Obstet. and Gyn. Brit. Emp.*, 1902, i, p. 138.

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degree. In my early days of performing Cæsarean section I had ether administered—of course by skilled anæsthetists—but found that the child was often asphyxiated and that hot and cold baths, stimulants and assistants were required to start respiration in the infant. The occurrence of asphyxia in the child is still common in the practice of many obstetricians. Dr. Hubert Roberts has pointed out its fairly constant occurrence.

For many years I have used the method about to be described and can assert that the Cæsarean child has never been asphyxiated by the anæsthetic thus administered.

The method is as follows: No preliminary hypodermic injection is given. The patient, prepared for operation, is placed in the recumbent posture on the operating table in the theatre and the anæsthetic is administered there. Operator and assistants are prepared and the towels adjusted. *Chloroform* is then administered by the anæsthetist, who informs the operator when the patient is ready. The operation is then rapidly performed and the child, usually delivered within thirty or forty seconds from the commencement of the incision, always breathes and cries at once. *Ether* is then administered for the rest of the operation, usually by the open method. If there is any unusual loss of blood a hypodermic injection of *puitrin* is given.

The result of this method of administration is that in all cases in which the classical Cæsarean section is done before or soon after the onset of labour the child is born free from asphyxia and cries at once.

I think it is the ideal method of administration; but I am anxious to have the opinion of members of the Section upon this point:—

Does the mother run any appreciable risk in having the operation performed while under chloroform administered by an expert anæsthetist? I think she does *not*, having particularly in mind the tolerance of parturient women for small doses of chloroform.

### DISCUSSION.

Mr. A. L. FLEMMING (President) read extracts from a letter from Dr. Miles Phillips, of Sheffield, in which the following sentences occurred:—

"It is just possible that babies do not cry out so quickly after chloroform as after ether. . . . I expect a good anæsthetist to keep the patient very lightly under (ether) after the abdomen is opened until I commence to sew it up again. . . . Since my anæsthetist has used Shipway's method I have never had to worry about the anesthesia, whereas formerly when chloroform was used I was always doing artificial respiration."

The President (resuming) suggested that discrepancies in results might be due as much to the degree of anesthesia as to the nature of anæsthetic employed, and said that it was worthy of note that Dr. Phillips made a point of light ether anesthesia being used during the essential part of the operation.

The President also read a letter from Dr. Goodman Levy, in which Dr. Levy said:—

"The immunity of women in labour from chloroform syncope is a remarkable thing, and it is difficult to say exactly how this immunity comes about, but I think the peculiar temperament of the pregnant woman has something to do with the matter; there is little or no excitement 'going under,' cardiac sympathetic impulses are probably moderated, and the heart itself must be presumed to be less irritable under these special circumstances."

Dr. LIONEL SMITH said that it was a well known fact that infants delivered by Cæsarean section were frequently cyanosed and difficult to revive. He had frequently delivered women naturally who were fully under an anæsthetic for long periods and yet the babies were not cyanosed and cried immediately on birth. He thought there was some other factor besides the anæsthetic giving rise to the cyanosis after Cæsarean section. It should be remembered that in Cæsarean section the child was "untimely ripped" from the uterus. Was it not probable that in its descent through the maternal passages during natural delivery, the child was stimulated and thus prepared for its separate existence?



Dr. KINGSFORD said he agreed with Dr. Spencer. Years ago, when the mothers were anæsthetized with ether, or CE, there was often difficulty in getting the child to breathe. At Dr. Spencer's suggestion he had since given only chloroform for the induction; at first with the Vernon-Harcourt inhaler, but as with this method the induction seemed, to Dr. Spencer, rather prolonged, he (Dr. Kingsford) had for some years used only a Schimmelbusch mask. About 2 dr. were poured on the mask, which was gradually lowered over the patient's face, and the administration was continued till the corneal reflex was almost abolished, when the signal for operation was given, and the chloroform withheld to watch the effect of the first incision. Induction time was about three to five minutes. As soon as Dr. Spencer appeared satisfied as regards possible hæmorrhage, ether was given, warm and moist, by a semi-open method. In about thirty cases, thus treated, the infant had always started breathing at once, while no anxiety had been experienced as regards the mother.

Dr. H. W. FEATHERSTONE (Birmingham) said that he had records of thirty-four cases of Cæsarean section anæsthetized by himself. These were principally the patients of Mr. Beckwith Whitehouse. In every case the child survived for at least twelve hours, and no less than three pairs of twins were delivered. On each occasion the uterus contracted well after delivery. Only one mother was lost. She had suffered from chronic bronchitis, three previous attacks of broncho-pneumonia, mitral stenosis, and excessive fatigue from prolonged labour. Twins were delivered, and the woman died of broncho-pneumonia on the ninth day. In every case induction was by ethyl chloride on an open mask, with ether for the maintenance of anæsthesia. No chloroform was employed. He (Dr. Featherstone) considered morphia to be harmful, but used a routine preliminary injection of atropine. In two cases repeated blood-pressure readings did not exhibit any marked alteration at the time of delivery of the child. He considered chloroform to be contra-indicated in exhausted patients, but had observed some signs of drugging by ether in one or two children. Evidently the child should be delivered as soon as possible after the commencement of anæsthesia.

Dr. BLOMFIELD asked whether Dr. Spencer attributed the asphyxial state of the babies when ether was used to the action of the drug on the infant's respiratory passages and excess of mucus, or to toxic action on the respiratory centre. If the latter was the cause it seemed strange that it should occur under ether rather than under chloroform, which was usually so much more prone to lead to this and cause the respiratory failure.

Mr. H. E. G. BOYLE said he was disappointed that no mention had been made of the more modern forms of anæsthesia, and especially that no reference had been made to the valuable work of Dr. Bourne of Montreal, who had shown that the results of giving gas oxygen to women in labour (and this included Cæsarean section) were infinitely better than when any other form of anæsthetic was used. When he was in America last year as the representative of the Section, he had himself seen Dr. Bourne administer gas oxygen to a case of Cæsarean section, and he had never witnessed a better anæsthetic for the purpose. He was surprised to hear Dr. Spencer say that he had more trouble with the breathing of the child when ether was given, and could only state that that had not been his personal experience. It was commonly said that women in labour could be given chloroform with impunity, but in his opinion that was a completely false statement, and he felt quite certain that if the real statistics of deaths from chloroform during childbirth could be obtained, members would be appalled at the high mortality. He knew that it would be extremely difficult for gas oxygen to be used extensively in country practice, but he thought that in London and other large centres where skilled anæsthetists were obtainable, more use of this valuable anæsthetic might be made in cases of Cæsarean section and labour.

Dr. W. J. MCCARDIE said that as regards the mother it was all-important that the uterus should rapidly contract after delivery, and that as regards the child anæsthesia should be as little toxic as possible, and last a minimal time. These requirements implied that only a method and dosage be employed which could be controlled absolutely, and be ended at any minute, and therefore ruled out any other means but inhalational anæsthesia by the least toxic drug possible. The only accessory drug he used was atropine  $\frac{1}{16}$  gr. In the early days of gas and oxygen anæsthesia he had used it several times with the largish doses of morphine and scopolamine then

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customary, but found that the child did not breathe freely for some time after delivery. He wondered what, if any, preliminary medication Dr. Bourne, of Toronto, used in his gas and oxygen cases. It would be instructive to have investigations of the infant's blood made after delivery under various methods of anæsthesia, and to compare the results with those obtained from an examination of the mother's blood.

Dr. SHIPWAY said he was opposed to the use of a routine method of anæsthesia for Cæsarean section. The condition of the patients varied so much that he thought it desirable to select the method for each individual case. In the past he had preferred to use ether (warm) and oxygen, preceded by CE for induction. He thought, however, that gas and oxygen was more suitable for many of these patients, particularly if the condition was poor or there was a state of shock. He had used this anæsthetic in two cases of this description with very satisfactory results, and intended to use it more frequently in the future. There was of course no difficulty in obtaining relaxation; and the effect of the gas upon the baby was very slight. He thought that when asphyxiation of the baby had followed the administration of ether, overdosage was almost certainly the cause.

Dr. C. F. HADFIELD, speaking from a fair experience of anæsthetics for this operation, said he felt obliged to disagree with Dr. Spencer regarding the condition of the baby's respiration following ether or chloroform. Inquiry from an obstetrical surgeon who had performed upwards of 150 Cæsarean sections had also elicited the opinion that infants breathed better when ether had been administered to the mother than when after she had taken chloroform. In the half dozen or so cases in which he had used spinal analgesia (stovaine) the results had all been most satisfactory. With regard to the statement of a French author that stovaine had the effect of stimulating the contractility of uterine muscle, Dr. Hadfield said that he had not been able to determine that the contractility was affected one way or another; what was the experience of other members? In the class of case in which Cæsarean section was only adopted as a final resort when various manipulative measures under more or less continuous and protracted chloroform anæsthesia had failed, the use of any further chloroform was to be avoided at all costs; and he quoted such a case in which death had resulted from delayed chloroform poisoning.

Miss D. C. LOGAN asked Dr. Spencer whether, when he used ether for Cæsarean section his technique was the same as described for his  $\text{CHCl}_3$  operations? Did Dr. Spencer lay special stress on the short time that the patient was anæsthetized before extraction of the infant? i.e., through having operators and patient all ready for operation before the anæsthesia was begun.

Dr. HERBERT SPENCER was disappointed that more obstetricians had not taken part in the discussion: had they done so he thought their experience would have been the same as Dr. Lionel Smith's, that Cæsarean section children were often asphyxiated. He could not agree that the operation itself induced the asphyxia; that view was disproved by his cases. He would point out that his patients were only fully under the influence of chloroform for about two minutes and were not to be compared with a patient mentioned by one of the speakers who had had it administered for twenty-four hours! Of the cases mentioned by the President and Dr. Hadfield nothing was said as to the time the patients were under chloroform before they were delivered. He had had no experience of ethyl chloride and ether; but he thought the fact that Dr. Featherstone used ethyl chloride suggested that he did not like ether alone; and it did not surprise him that he found "signs of drugging by ether in one or two children." Dr. Spencer did not think the asphyxia was due to mucus in the air passages. He was aware that gas and oxygen were very popular on the American Continent. It was an excellent method of anæsthesia which he had often had administered to gynaecological patients; but, in his opinion, it was not ideal for Cæsarean section, as it did not render the patient as calm or as quiet as chloroform. There was no doubt that morphia was a very dangerous drug for the child. He had less objection to atropine, though he did not value it highly. In reply to Miss Logan, Dr. Spencer said he *did* lay great stress on the shortness of the anæsthesia and its administration in the theatre. His ether cases had been anæsthetized in an adjacent room, which entailed a longer anæsthesia. He did not know whether the children would have been free from asphyxia under ether administered for an equally short time; but his preference for chloroform till the child had been delivered would still remain, since parturient patients took that anæsthetic with readiness and even avidity, which was far from being the case with ether.

## Section of Anæsthetics.

President — Dr. A. L. FLEMMING.

### Case of Cardiac Arrest under an Anæsthetic followed by Heart Massage.

By E. STANLEY ROWBOTHAM.

SERGEANT F., aged 43. Wounded September 27, 1918, bullet entering at left nasolabial fold. Removed April 11, 1919, from region of right coronoid process. November, 1922: Complains of discharge from left side of nose, and frontal headache. Spur to right of septum and septum very crowded. Middle turbinates swollen. Operation for resection of septum (not commenced).

Patient was rather a weedy-looking man of small build. Examination showed heart normal in size, no murmurs, pulse regular and full. Chest expansion somewhat limited. Nothing abnormal found in urine. Eyes somewhat prominent, but no signs of Graves' disease present. Patient was very nervous in spite of the fact that he had had a hypodermic injection of morphine sulphate  $\frac{1}{4}$  gr., hyoscine hydrobromide  $\frac{1}{100}$  gr., atropine sulphate  $\frac{1}{100}$  gr., half an hour before being brought into the theatre.

Commencing at 10.15 a.m., the nose was first sprayed with cocaine hydrochloride 20 per cent., adrenalin chloride 1 in 1,000 (in equal parts), and then packed twice with gauze soaked in cocaine hydrochloride 20 per cent. (three parts), adrenalin chloride 1 in 1,000 (one part). Administration of the anæsthetic was begun at 10.45 a.m. when the patient appeared normal and spoke rationally, although still nervous. Induction by ethyl chloride-ether on an open mask, 8 c.c. of ethyl chloride and 5 oz. of ether being used. No chloroform was given during the whole of the administration. The patient went under easily with no struggling. A tracheal catheter was passed immediately relaxation of the jaw was obtained, and air and ether mixture administered intratracheally. A sponge was placed in the post-nasal space. At this period the patient was breathing regularly, was of a good colour, and appeared to be taking the anæsthetic well.

Without any warning, about ten minutes after the commencement of induction, and before the surgeon had started to operate, breathing stopped, the pupils became suddenly fully dilated (they were almost pin-point a minute before), and the pulse was found to be absent. The ether was stopped at once and oxygen run through the intratracheal apparatus. Artificial respiration by chest compression was also commenced, but with no result. Within five minutes of the cessation of breathing Mr. Kilner opened the abdomen, incised the diaphragm, and massaged the heart through the left pleural cavity. Artificial respiration by chest compression was continued and the limbs were

bandaged. The heart very quickly responded to massage, but after a few beats became tense, filled and unresponsive. The method of artificial respiration was now changed to that of Silvester and the heart was at once easily emptied of its contained blood, and was very soon beating strongly and regularly, until at the end of five minutes the pulse was regular and full at 110. As the heart gained strength the patient gradually began to make respiratory efforts, and was breathing 30 to the minute six minutes after the first attempt at cardiac massage. Strychnine sulphate  $\frac{1}{30}$  gr. was now given hypodermically.

The diaphragm was sutured and the abdomen closed, and the patient returned to bed at 1.15 p.m. His pulse and respirations, however, gradually rose (at 6.15 p.m. they were 150 and 48, temperature  $101.2^{\circ}$  F.), he continually coughed up frothy blood and mucus, and required constant administration of oxygen to keep him of a good colour. He was given caffeine, digitalin, and atropine hourly, but he never regained consciousness. A condition of acute pulmonary œdema set in, and he died at 3.25 a.m.

There appear to be three points worthy of note in this case:—

(1) The fact that Silvester's method of artificial respiration appeared to be a very much more efficient aid to the heart than simple chest compression.

(2) This was a death under ether. No chloroform whatever was used, and only a very small amount of ethyl chloride at the commencement of induction.

(3) The question as to whether the cocaine with which the patient's nose was packed had anything to do with his heart failure. The gauze was wrung dry and certainly no cocaine was swallowed.

Mr. T. POMFRET KILNER said that his interest in the case described by Dr. Rowbotham had arisen by chance. He had happened to call in at the operating theatre just as difficulties with the patient arose, and, as he was standing by to help, he was requested by the surgeon in charge of the case to perform cardiac massage when it was found that the heart had stopped beating. He (Mr. Kilner) opened the abdominal cavity by a mid-line incision above the umbilicus and, slipping his left hand under the costal margin and raising it well, exposed the anterior attachment of the diaphragm. He made a small opening in the latter near to its costal attachment and a little to the left of the mid-line and through this reached the heart. The heart was flabby and toneless. With two fingers behind and the thumb in front, he compressed and relaxed two or three times at a rate estimated as that of the normal heart-beat. A few weak but spontaneous beats were started in this way; the heart stopped again, but was now felt to be tensely filled with blood. Continued efforts at compression and relaxation failed to empty it, and the beat did not start again until artificial respiration, by carrying the arms well above the head, was performed. The heart almost immediately emptied and a steady, strong, rhythmical beat became established. After waiting for a little while, the opening in the diaphragm was closed by catgut sutures and the opening in the abdominal wall repaired.

The progress of the case from this point onwards had been described by Dr. Rowbotham. He (Mr. Kilner) had performed the post-mortem examination of the case, but had found very little to account for the primary collapse of the patient. The following appearances were noted:—

*Lungs*.—Old healed tuberculosis, left apex. More recent and more extensive tuberculosis with formation of a large cavity, right apex. Both apices surrounded by dense pleural adhesions.

*Heart*.—Normal in size and appearance. Muscle normal in appearance on section. Small patches of atheroma on septal cusp of mitral valve. Fairly extensive patches of atheroma in aorta. There was a large clot of ante-mortem type in the right ventricle. He (Mr. Kilner) suggested that this clot was formed, or started forming, while the heart's action was in abeyance and that it might be looked upon as responsible for the failure of the cardiac massage and finally for the patient's death.

It was in the hope that one might hear helpful discussion which would guide one to perform cardiac massage at the most opportune moment and by the best method that this case had been brought forward and described in some detail.

The case was further discussed by the Members.

Mr. KILNER (in answer to criticism of method employed) said that the opening in the diaphragm had been made at the express wish of the surgeon in charge of the case. In the only other case in which he had had to perform cardiac massage no opening was made in the diaphragm. The heart beat was restored quite satisfactorily but the patient died about twenty-four hours later.

## The Effects of Vagal Trauma on the Anæsthetized Patient.

By C. L. HEWER, M.B.

THE two cases which are described below both illustrate the effects of surgical interference with the vagus nerve, a subject which concerns the anæsthetist closely.

*Case I.*—Patient, a male, aged about 60, upon whom a month previously a laryngectomy had been performed for extrinsic carcinoma. He was breathing through a tracheotomy tube, and the present operation was for the excision of malignant glands from both sides of his neck. He was anæsthetized with chloroform on a Hahn's cone connected to his tracheotomy tube. The anæsthesia was quite smooth and normal for the first part of the operation on the left side of the neck. About half way through the opposite side, however, the surgeon cut a large vessel, and hastily applied an artery forceps. The patient instantly stopped breathing and turned a greyish colour. I could not detect any radial pulse, and the capillary circulation had apparently ceased. On explaining the situation to the surgeon, he removed the forceps, and the colour of the patient's lips changed almost at once from grey to blue. His pulse again became palpable, feeble at first and then stronger, but extremely slow in rate—about 20 beats per minute. Respiration was also re-established gradually and his colour returned to pink. The operation was finished somewhat hastily, and the patient was put back to bed. He did not regain consciousness for three days, and during that period his pulse never exceeded 40 beats per minute, and he exhibited the explosive type of cerebral vomiting. At the end of two weeks he was comparatively normal, and left the nursing home a few days later with a pulse of 60. I do not know the subsequent history of the case. On talking the matter over afterwards with the surgeon, he said that he was dissecting behind the carotid sheath at the time, and considered it quite likely that the forceps were actually on the vagus nerve itself.

*Case II.*—Patient, a girl, aged 20, who had had many previous operations on a chronic left-sided empyema with collapse of the lung. It was decided that a decortication of the left lung should be performed. She was induced with ethanesal in a Clover's inhaler, and anæsthesia was maintained by gas and oxygen given through an endotracheal catheter. There was no difficulty at all with the anæsthetic, and the operation was begun by making a large osteo-plastic flap on the left side of the chest. When the pleural cavity was opened I slightly increased the intrapulmonary pressure, so that it oscillated between 10 and 15 mm. Hg. The thickened pleura was peeled off the collapsed lung, and all went well until the portion near the posterior part of the root of the lung was being reflected. At this point the patient suddenly

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stopped breathing and became quite pale. I immediately asked the surgeon to feel her heart, which he did through the wound, and he reported that it was quite flaccid, and was not beating or fibrillating. Cardiac massage was at once begun, and the lungs were intermittently inflated with oxygen through the catheter. That an efficient circulation was established was shown by the return of a pink colour to the mucous membranes, but the heart gave no response, and the patient died.

*Post Mortem.*—The course of the left vagus was dissected out, and the nerve was seen to end in the pleura, which had been reflected posterior to the root of the lung. It is therefore almost certain that the main trunk had either been actually divided or that direct trauma had been suffered by it.

In addition to these two cases, I have been told of another by a surgeon who was dissecting out malignant glands from behind the carotid sheath. This patient was in good condition, but died quite suddenly on the assistant retracting the sheath somewhat forcibly. I regret that I do not know what anæsthetic was employed in this case.

In a recent number of the *American Journal of Surgery*, there appeared an account of a patient who had been operated upon, and was being transferred from the table to a trolley. His head rolled over so that his neck pressed against the anæsthetist's arm. This patient suddenly collapsed, and the reason put forward was that his vagus nerve was compressed against the transverse process of one of his cervical vertebrae.

With reference to the physiology of this subject, the inhibitory action of the vagus nerve on the heart was discovered by the brothers E. H. and E. F. Weber in 1845.

The first instance of a fatal case in man was described by Thanhooffer in 1875, who tells of a case in which both vagi were compressed in the neck, a procedure which was followed by loss of consciousness and stoppage of the heart.

In animals, section of both vagi causes the respiration to become slower and deeper, and as the tonic inhibition of the heart is removed, the heart beats more rapidly.

It is necessary, however, to bear in mind that the nervous mechanism controlling the heart is much more complex in man than in the lower mammals, and that the human heart is much more intolerant of rhythmic disturbances. This is well seen in the many experiments which have been made on animals suffering from primary cardiac failure under chloroform. In practically every case it is possible to restart their hearts by cardiac massage, but unfortunately the same does not hold good in man. It therefore seems reasonable to suppose that the stimulation or section of one vagus is far more likely to produce cardiac arrest in man than it is in animals.

A further difference is to be observed in the action of atropine. An adequate dose of this drug injected into animals stops all these vagal effects, but it should be noticed that in both the cases related above there was a preliminary injection of 100 gr. atropine.

With regard to the exact nature of the heart stoppage, the vagal fibres are only distributed to the auricles of the mammalian heart and not to the ventricles. Consequently, when the vagus is stimulated, the sino-auricular nodes and the auricles are directly affected, and the ventricles only cease beating because they no longer receive their normal stimuli. The whole heart, therefore, stops in diastole. Sometimes, however, if the stimulus to the vagus is prolonged, the



heart may again begin beating slowly. This is known as vagus escape, and is due to the ventricles contracting at their own independent rate. This phenomenon apparently occurred in the first case described.

A short time ago it was suggested that the right and left vagus had different functions. In some of the lower vertebrates, especially the terrapin, all the inhibitory fibres are contained in the right vagus, but in the higher mammals and in man there is no doubt that stimulation of either vagus may produce cardiac inhibition. In the first case described the right vagus was involved, and in the second the left.

In addition to the direct stimuli to the vagus which have been described, there is little doubt that reflex stimulation may occur. Thus, if we accept the theory that the first action of chloroform is to render the vagus centre hypersensitive, there is no difficulty in accounting for the deaths which occur on beginning an operation under light chloroform anæsthesia.

There is one other aspect of the subject which is interesting, and that concerns dissection of cervical glands in children. I think that most anæsthetists will agree that of all the operations in children, this is the one most frequently followed by vomiting and collapse. It appears possible that traction on the vagi may be the cause. It would be interesting to hear the opinions of other anæsthetists on the subject.

### Cases of Difficulties due to Important Points having been missed at the Preliminary Examination.

By A. I. FLEMMING (President).

THE following cases illustrate the importance of careful routine examination of patients preceding the administration of anæsthetics:—

*Case I.*—Schoolboy, aged 12. Operation for small lipoma of thigh. Auscultation revealed a small area with crepitations at apex of right lower lobe of lung. Operation was abandoned. The patient died nine months later from phthisis. Had an anæsthetic been administered it would undoubtedly have been considered the cause of the lung affection.

*Case II.*—Male, aged 69, with temperature 102° F. and head pain. Operation for suppurative frontal sinusitis. Examination revealed a small patch of pneumonia. No operation was performed. Twenty-four hours later well marked pneumonia had developed.

*Case III.*—Male, aged 60. Operated on under open ether anæsthesia for extraction of teeth. Patient complained of frontal headache and his complexion was sallow. Twenty-four hours after operation pneumonia had set in. On subsequent inquiry it transpired that the patient had felt shivery and had been suffering from malaise for two days before the operation. A more careful overhaul might obviously have revealed the presence of lung trouble and led to the operation being abandoned.

*Case IV.*—Female, aged 50. Operation for extraction of teeth under nitrous oxide at a dentist's surgery. Patient appeared to be very nervous and frightened, and her condition grew more alarming instead of improving with the loss of consciousness. Administration was stopped and no operation performed. Subsequently it turned out that she was an advanced case of exophthalmic goitre,



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with very little enlargement of the thyroid at the time. Had the condition been recognized administration would certainly have been delayed until the symptoms of fright had subsided. Six months later this patient took nitrous oxide without any untoward incident.

*Case V.*—Schoolboy, aged 17. Operated upon for acute appendicitis. The patient had been under careful observation for seven days—at the time of operation the temperature was 100° F. A tendency to retch caused difficulty and necessitated somewhat deeper anæsthesia than is usual in such cases. Before closure of wound he was allowed to vomit and subsequently relaxation was more satisfactory—but cyanosis, especially of the lips—led to the suspicion that vomited material had been aspirated. After operation cyanosis persisted and was accompanied by weak rapid pulse—144 per minute, and a respiration rate of 40 per minute. The mucous membrane of the mouth was remarkably dry and it was more than an hour before the boy could be induced to cough or vomit. After coughing the symptoms immediately improved, but the improvement was maintained only for thirty minutes, after which the rapid pulse and respiration returned with cyanosis. Twelve hours later pneumonia was diagnosed but twenty-four hours later well marked measles had developed and there was no further lung or heart trouble. Had the patient died before the diagnosis of measles had been established the death would have been attributed to aspiration pneumonia or collapse of lung.

## Section of Anæsthetics.

President — Dr. A. L. FLEMMING.

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### General Anæsthesia in Dental Surgery.

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I BELIEVE that the subject of general anæsthesia in dental surgery has not been dealt with previously at any meeting of this Section or of the old Society of Anæsthetists.

The position of the anæsthetist in dental surgery differs from that in most departments of general surgery from the fact that the operation involves the mouth, and, consequently, respiration. Again, the anæsthetist, in this country at any rate, not only administers the anæsthetic but is also actively engaged in helping the operator, is usually the only medical man present, and sometimes the only one competent to deal with vital emergency. Patients come up usually without any very special preparation, mentally alert, active in body, and brains not obscured by sedatives, unless administered by themselves. "They are clothed and in their right minds." Often they are intensely nervous and critical of their surroundings and of all things, even to the smallest detail. They do not expect a rough time; they think "only a tooth or a few teeth are to be extracted under gas." On the other hand, they may be worn out by hard work or society functions.

In general surgery it is the rule, if reasonably possible, to do all that is necessary at one sitting; in dental surgery I think that the same rule should hold good. It is, I gather from reading, sometimes advised that the extraction of a few teeth at a time is the best practice. I thoroughly believe in making it possible for the operator to do what he wishes at one sitting and to encourage him to do so. The late Sir Frederic Hewitt spoke of the "progressive insusceptibility" of patients to nitrous oxide when given at intervals of one to ten days. This condition may be partly physical but is certainly chiefly mental, and I think that if we were to ask the patient he would, in ninety-nine cases out of a hundred, prefer that the operation be finished once and for all. Therefore it is my rule that what the dentist proposes to do should be done, even if it involve the administration of a major anæsthetic in his room and some immediate discomfort and inconvenience to the patient; otherwise the patient will be dissatisfied and will think that he could not take the anæsthetic or that the operator could not operate. Luckily he very rarely thinks that the anæsthetist could not administer the anæsthetic.

There is a difference between extractions as performed years ago and nowadays. Formerly teeth were usually taken out for pain or local disease,

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and sometimes more or less roughly, because speed mattered under the conditions of anæsthesia then prevailing. Now dental surgery is associated with the treatment of general diseases and especially toxic diseases, as well as local disease, and many tough, brittle, root-filled and crowned teeth have to be very carefully extracted with as little local damage as possible. This means that prolonged anæsthesia is necessary.

When I first thought of this paper I estimated my cases to be very much more numerous than they really are. My own figures for dental anæsthetics from January, 1898, to the present date are 18,338. In the early days one gave ordinary gas, then prolonged gas by means of a mouth-tube; later the modern prolonged method, so admirably worked out by Paterson, was used. In January, 1901, I began to use ethyl chloride as an intermediate anæsthetic between gas and ether. Then, as now, I regarded nitrous oxide and ether as our main anæsthetics in dental work. I have had no fatality during anæsthesia, although one occurred a few hours after as will be described later, and have only once taken out my tracheotomy case, which, fortunately, it was unnecessary to use. My longest purely gas anæsthesia lasted thirty-three minutes, and my oldest patient, a man aged 94, had gas and oxygen. Dangerous collapse, necessitating formal artificial respiration, occurred in three cases, once during chloroform and twice during gas anæsthesia. One case in twenty-three, i.e., 794, had a major anæsthetic, that is to say, ether and/or chloroform solely or mainly. Nitrous oxide, ethyl chloride and somnoform I regard as minor anæsthetics.

The total number of anæsthetics administered at the Birmingham Dental Hospital from October, 1896, to September, 1922, is 91,734. Of these about 3,500 were ethyl chloride, about 200 ether, leaving about 88,000 gas cases. In these no oxygen was administered. There have been no deaths recorded during or after anæsthesia; in fact, there has never been a fatality or even tracheotomy in the history of the hospital, and that is going back more than forty years. Of course it is rarely possible to obtain information as to after-effects such as shock due to anæsthesia or trauma in hospital patients. In a large proportion of the gas cases anæsthesia has been carried out by students under supervision. Prolonged gas without oxygen is the routine anæsthetic.

These figures tend to show the safety of gas given by short or prolonged methods for all sorts and conditions of patients, when administered by anæsthetists, house-surgeons and students in a dental hospital. Adding the hospital and my own figures we have more than 100,000 administrations of gas alone, without fatality. The proportion of major anæsthetics in hospital figures is about 1 in 36. Thus the proportion of major anæsthetics to minor anæsthetics in my series is larger than in the hospital series. The difference may be explained by the fact that when I gave an anæsthetic I always tried to help the dentist to finish his operation at one sitting, whereas in hospital work it would seem that this principle cannot be so generally carried out. Hospital patients appear to bear prolonged operation under gas better than do private patients. It is usually impossible to administer major anæsthetics in the dental clinic because of the difficulty of after-treatment and removal. As in the hospital cases, so in my own; in the vast majority of patients gas alone has been used. In fact, I have only administered gas, and oxygen in special cases.

In hospital our students are taught to administer nitrous oxide and no other anæsthetic. The signs of anæsthesia under gas, or more strictly speaking, the signs of asphyxia, are definite and well recognized, and unless gas and

oxygen be really well administered—no easy matter—it is of comparatively little value.

My own figures are small in comparison with those of other administrators. Hewitt, for instance, administered gas and oxygen in at least 20,000 cases. Ream, in America, states that he has, "during eighteen years performed approximately 30,000 administrations of gas and oxygen without a single accident." "One office in Philadelphia reports nearly 300,000 administrations without an accident." Teter, years ago, stated that he had given gas and oxygen in 13,000 cases. Recently he sent out a questionnaire to extracting specialists, and from their answers reported that:—

Nitrous oxide was administered alone in	...	...	...	938,924 cases
Nitrous oxide with air in	...	...	...	32,172 "
Gas and oxygen in	...	...	...	190,724 "
				1,161,820

There were two fatalities in this series.

It would be interesting to have the collective statistics of the various dental hospitals in Great Britain for a series of years.

The choice of the anæsthetic is associated with the type of patient, and the duration of the operation. As a general rule I choose nitrous oxide without, or, sometimes with, a very small amount of ether added, as I shall describe later. To patients suffering from serious lung or heart troubles or toxic diseases I administer gas and oxygen if possible. Big and difficult cases I prefer to anæsthetize in a nursing home. During the War experience showed that nitrous oxide was of no use for soldiers, and I treated them all as major surgical cases. Small children are a difficult problem. Some years ago I had to anæsthetize some hundreds of them sent up, in batches at a time, from a Board School. They were brought up by their teachers and were under perfect control. They rarely made any fuss and did as they were told. I found that a prolonged administration of gas and oxygen for the smaller children gave me excellent results. Ethyl chloride upset them too much. In private work a small child is often uncontrollable; consequently one will fail with gas. In such a case I administer ethyl chloride and ether, in mixture or in sequence, from Ormsby's inhaler. Fortunately one finds that if patients, children and adults alike, bear gas badly, they are very little, if at all, upset by a major anæsthetic. Oldish, greyish and worn people feel deprivation of oxygen seriously. Occasionally in them I have noticed that gas without oxygen for a few minutes, especially if there has been any marked asphyxial element, has caused pain in the chest and over the heart for a week or two afterwards, indicating some degree of cardiac strain and possibly dilatation. It is well to bear this in mind. Crouch, some twenty-five years ago at a meeting of the Society of Anæsthetists, recorded two cases of dilatation of the heart during anæsthesia. The first patient, a nurse, was given gas and air for a short operation on a septic arm; the apex beat during administration moved from well within the nipple line to 2 in. outside it. In the second case gas and oxygen was administered to a girl who had a septic leg. Though her colour was perfect her heart rapidly dilated. This case shows the striking fact that in the absence of cyanosis there may be dilatation of the heart. On the other hand Paterson recorded a long gas and oxygen surgical case, lasting two hours twenty-seven minutes (varicose veins), in which the heart was carefully watched and showed no cardiac dilatation.

The choice of the anæsthetic has been suggested to me more than once by physicians, who, I think, exceed their function in so doing. Moreover, I have

known a physician forbid a general anæsthetic, even gas and oxygen, to a patient for whom his doctor and I advised it. The choice of the anæsthetic properly rests with the anæsthetist. Again, only the other day a patient was seen by a dentist and myself, and we agreed that she was perfectly fit to take a major anæsthetic for the extraction of about seventeen teeth. She was not toxic, was very frightened, came from the country at much inconvenience, and with reluctance promised to go into a nursing home to have the teeth extracted at one operation. Then she saw a physician who told her that for fear of lighting up infection the extractions must be done at two sittings. In the face of this we could not urge our advice. The patient has had half her teeth extracted and is being unnecessarily put to a great deal of inconvenience, expense, and mental worry, and has not been seen since. The position is unfair to her and to us.

It is stated that gas and oxygen does not appear to be altogether safe, for Warner<sup>1</sup> and other authorities assert that a perfectly normal colour can be maintained while the patient is presenting all the symptoms of asphyxia, including convulsions. There still seems to be doubt in the minds of some dentists as to the safety of ether as compared with that of gas. I am confident that ether is just as safe and even safer than gas in dental surgery.

A propos of this, in a discussion on open ether<sup>2</sup> Bellamy Gardner stated that he had "a patient with mitral regurgitation, who, requiring removal of some exostosed teeth, developed a most alarming syncope on two occasions under nitrous oxide and oxygen in the dentist's chair. On a third occasion six difficult extractions were accomplished in the upright position under open ether without faintness or subsequent vomiting." I feel sure that in many very prolonged cases, however successful, the substitution of ether would result in less shock to the patient. Shock is generally felt more when the patient has gone home and would not have occurred if the patient had been put straight to bed.

In the ordinary prolonged method of administration gas is mixed to a certain extent with air in the mouth, and one does not get the degree of asphyxia possible in administration for general surgical work. It would be interesting to know how much partial—for it must nearly always be partial—asphyxia raises the blood-pressure in dental anæsthesia. I was suddenly called in one evening to administer gas to a man, aged about 55, for extraction of two teeth. He was short, healthy-looking, of ordinary physique and very cheerful disposition, making fun of the proceedings. He had had gas before. I administered prolonged gas for about one to two minutes. There was just very slight change of colour due to the ordinary degree of respiratory obstruction during extraction of a couple of lower teeth which took about two minutes at most. The patient quickly recovered, laughed and made a joke, and proceeded to wash out his mouth. While I was packing up my apparatus he dropped his glass. He was given another, which he took, but did not use. He sat quite still in the chair and never spoke another word. His right side was found to be paralysed. Coma and paralysis increased. He was removed to a nursing home and died five hours after this attack. His doctor told us that he had formerly treated the patient for high blood-pressure, but had not seen him for the last nine months. No post-mortem examination was held. I wonder if the addition of oxygen to gas would have made any difference.

<sup>1</sup> *Journ. Amer. Med. Assoc.*, December 4, 1915.

<sup>2</sup> *Brit. Med. Journ.*, September 17, 1910.

Complaints have been made to me, by more than one dentist, of collapse of the patient during an illness after gas administration, and I have been asked whether this might be due to impurity of gas. On examination none has been found, and the makers disclaim anything wrong. I do not know of impurities occurring in gas in this country, but Teter, in 1912, had reported to him within a month two fatal cases in which nitrous oxide and oxygen were used, death being due to impure gas. There were such peculiar symptoms manifested in these cases that the cause could not be overlooked. The patients had a very peculiar greenish-red colour and the blood seemed almost of a rusty hue. Collins also reported a fatal case with these symptoms, and two other cases in which the gases were impure. His patients were quite sick for a day or two, but recovered. In my own experience I have had no reason to suspect nitrous oxide.

Patients in most cases being out-patients little can be done in the way of preliminary sedative treatment. For very nervous people a full dose of bromide overnight, and another dose a few hours before anæsthesia, are very useful. Ten grains of chloretone will sometimes do much good. Aspirin is not advisable as it tends to cause cyanosis unless ether be the main anæsthetic. Given immediately before anæsthesia brandy or whisky are the only sure sedatives. If administered neat and freely they will sometimes work wonders, even in alcoholics. For instance a man, aged 30, who said that he had had gas before and could not be put under, drank twelve brandies and sodas before the operation. Anæsthesia by the prolonged method was perfect and was continued between two and three minutes. He had very pleasant dreams. Evidently one must not stint the quantity. I rely upon bromide and brandy in the case of frightened patients. Bilious subjects, who are usually sick after anæsthesia, should, if possible, be dieted and dosed with alkali beforehand.

For administration I use a modified Trewby's apparatus, and occasionally Paterson's. In the nose-piece of Paterson's apparatus I have had a hole bored, which serves as an expiratory valve; I cover it with my finger when necessary. In Trewby's apparatus the mouth-piece is exchanged for a metal form of Paterson's mouth-piece. Both mouth-piece and nose-piece are fitted with air-cushions, because the bare metal sometimes causes bruising and occasions bitter complaint on the part of the patient. I usually allow the operator to begin extraction of lower teeth after the first snore, and of upper teeth after the third snore. Sponges placed well forward in the mouth are used frequently. They prevent entry of air, help coagulation of blood by contact and absorb it, and serve as a useful vehicle for administering small quantities of ether, ethyl chloride or even chloroform on occasion.

A dental surgeon in Birmingham, Mr. Vaughan Tomey, some years ago showed me how he frequently administered a little ether on a sponge during gas anæsthesia. Ether is dropped on the sponge, which is then lightly wrung out and placed close to hand. If anæsthesia is not deep enough, the sponge is placed in the mouth and changed when necessary. In this way, the etherized sponge or sponges will often enable one to get through without resorting to a full dose of ethyl chloride or ether. Moreover, if the patient becomes slightly blue, or grey, a little ether acts as an admirable stimulant, and the patient's colour will improve. In fact, in many cases where there is no difficulty in maintaining anæsthesia, and if I wish to stimulate the patient, I use an etherized sponge. It gives a gas and oxygen effect. The patient rarely complains of the taste of ether. If I fail to control the patient I do not hesitate to pick up my Ormsby inhaler and to administer ethyl-chloride mixed



with or followed by ether. The Ormsby is far better than the Clover for dental work for it will stand being knocked about, breathing from it is very free and an unlimited quantity of ether can be poured into it and the deepest anæsthesia very quickly obtained. The sponges I use in the mouth are of two sizes. They are twopenny and threepenny toilet sponges sold on a card and are very coarse in texture. The etherized sponge has been of the greatest help to me. Small gauze-covered swabs are dangerous. After operation a sponge or two should be left in the mouth for a short time and rinsing should be discouraged as far as possible. It arouses the patient and delays coagulation of blood. The best after-stimulant is whisky or brandy. These are the only restoratives I use.

In connexion with Paterson's apparatus when induction is difficult, or fails, I have frequently used what I call a mouth-Ormsby, that is, a small bag inhaler with metal mouth-piece, into which I spray some ethyl chloride or ethyl, and apply to the mouth, at the same time using the nose-piece. This mouth-Ormsby has not infrequently saved me from failure.

For small or obstreperous children or stupid patients who struggle or will not breathe properly, I do not hesitate to apply the Ormsby straight away. If the operation be very short, as for extraction for a few teeth or for dealing with the frenum, rather than use ethyl chloride alone, I prefer to use ethyl chloride and ether in mixture or sequence. Ether causes much less after-effect than does ethyl chloride, though the latter is, of course, best for induction of anæsthesia. It is astonishing how little after effect is produced by inhalation of ether and how seldom the patient complains of unpleasantness. There is no doubt that it is far better to administer ether than to conduct a rather unsatisfactory prolonged gas anæsthesia. The shock is infinitely less; moreover, in a difficult extraction or series of extractions there is less bleeding during etherization, and the operator has a better chance. Occasionally the patient will object to any nasal or mouth inhaler whatever, then perhaps he will not mind a little ethyl chloride sprayed on some gauze or a swab held over his mouth, and one may gradually induce light anæsthesia without difficulty. If ethyl chloride be administered from an Ormsby only a few cubic centimetres should be sprayed into the bag and a sponge then lightly packed so as more or less to shut off the bag. Thus, too strong a vapour will not be inhaled at first.

In the early days of ethyl-chloride anæsthesia I used mixtures of it in 10 per cent. and 20 per cent. of alcohol, and also tried it when dissolved in the same percentages of white oil. I found that evaporation was slower and excitation less. The addition of 5 per cent. or 25 per cent. of bromide increased after-sickness.

In major cases I make it a rule, after having deeply etherized the patient, to continue with light chloroform anæsthesia by means of a mask over the nose or by one or two large nasal tubes. The obstruction to mouth-breathing caused by the dentist's manipulations, sometimes also by a sponge, usually produces nasal respiration. Occasionally I have used Crile's tubes or pumped a little chloroform through the nose into the mouth.

In Edinburgh the single dose method of Guy and Ross is advocated and taught to all students for routine work. A mixture of gas with ethyl chloride, or ether, or both, to which oxygen is added, is administered. It has the disadvantage that the anæsthesia is unnecessarily deep at first and may be more than enough, or, on the other hand, less than enough for the particular operation. According to Ross, nasal methods are the domain of the expert.

In my opinion, however, it is safer to teach the student to administer gas, and gas only, rather than a mixture of anæsthetics administered by routine dose and time.

Gwathmey's method, as used by Ecker in New York, of giving gas and oxygen which is often passed over a little paraldehyde through a sight feed machine, I have not used in dental work.

The often recurring trouble of respiratory obstruction, caused by depression of the jaw and pushing back the tongue in operation on the lower teeth, may sometimes be overcome by the use of a mouth tube, preferably one made of rubber.

If there be evidence of suffering during anæsthesia under gas, or gas mixed with ethyl chloride or ether, especially at the end of administration, it is a wise plan, in order to obliterate bad impressions, to continue administration of gas for half a minute or so after the operation is finished, and to suggest emphatically to the patient that he has done very well. It is important, too, that during awakening the patient should not see a bright light in front of him because bright sunlight or electric light will hasten return of consciousness and sometimes produce excitement. In fact in single dose administration of gas Dr. Downes, of Ludlow, tells me that by placing one's hand over the patient's eyes anæsthesia is prolonged for quite an appreciable period.

My experience of analgesia for painful dental procedures is very small. In my part of the country all that remains of this much vaunted method is the tapping of the spittoon to direct the patient's attention to the proper place.

One meets with difficult cases; for instance, what is one to do with a patient who habitually vomits after all anæsthetics, including gas, and who is "livery" after excitement? Such patients are generally of fair complexion and delicate. Again, what is one to do with a powerful man, aged about 35, who needs twelve difficult extractions, who is extremely nervous, and who retches and vomits if his mouth be examined. This man had previously given trouble to his dentist who thought he might have to do a tracheotomy after something had been done to the teeth. I ordered an injection of omnipon  $\frac{3}{4}$  gr. and scopolamine  $\frac{1}{16}$  gr. two hours before operation and administered chloroform in bed. He went to sleep lightly; then I twice tried to administer ether openly, but he coughed and became troublesome, so I had to continue with the chloroform. The anæsthetic and operation lasted about forty minutes and there were no after-effects. Again, a woman, aged 40, suffered from pernicious anæmia. Her red blood cells were  $1\frac{1}{2}$  millions and she had nearly died a week or two previously. Fifteen to twenty extractions were necessary. Anæsthesia was easily induced and maintained by ether and recovery was excellent. Occasionally one meets with patients who complain immediately before anæsthesia of heartburn or pain over the heart which is due to nervous flatulence. It sometimes first appears when the patient is seated in the chair and usually occurs in people who have few teeth left, especially women who drink much tea. A draught of neat spirit will often dispel the flatulence. If the condition be not treated there may be some collapse after anæsthesia.

I have only once seen syncope during anæsthesia. It occurred in a flabby, lymphatic young man to whom, in the dental chair, I administered gas and ether to light anæsthesia and followed them up with chloroform after a few minutes for some difficult extractions. The patient absolutely collapsed during further extraction; respiration and circulation stopped. I lifted him on to the floor and performed artificial respiration. When he was safe again, I induced the operator to finish the extractions, while the patient lay on the floor. Had



I fully charged up the patient with ether syncope probably could not have occurred. Three times I have observed collapse after anæsthesia in which there had been marked asphyxia, probably acute cardiac dilatation had occurred. I have no record of any case in which epilepsy supervened, although I have anæsthetized scores of epileptics, in fact I take no notice of epilepsy. Neither has there been any ill-effect from anæsthesia in pregnant women. Mania occurred in two cases after anæsthesia and operation under gas. In one the patient, an hysterical young Frenchwoman, became frenzied a few hours after, ran out of hospital into the street and was found by the police praying, and accosting people. She had to be taken to an asylum. The other instance was that of a man who had had gas some years ago, and afterwards used to wake up at night and "half throttle" his wife. Twice recently there had been two failures with gas inhalation. He was terribly frightened and "jumpy." I administered ethyl chloride followed by ether in the dental chair, soon got him well under and there was a perfect anæsthesia with recovery. After I left, the patient became very excited and walked up and down the passage with an attendant. He afterwards became quite maniacal and had to be treated and restrained for several days.

One patient, after gas, developed sharp and painful cramps in the abdomen and chest which, in a minute or two, "shifted down to the legs." The spasms lasted about five minutes.

Tetanic contractions of the limbs and rigidity of the body and shivering occurred in another patient on two occasions. These were probably hysterical in origin.

*Teeth in Air-passages.*—On two occasions teeth have slipped into or about the larynx.

*Case I.*—Patient was a dentist. Violent cough and spasm ensued for a few minutes. When he was bent forward and slapped on the back he coughed up the tooth.

*Case II.*—Patient, a big strong ex-soldier, who had a difficult extraction under gas, during which he struggled a good deal and threw back his head. The tooth was extracted but we could not find it. He immediately developed a very slight cough. After waiting for some time he was allowed to depart and was told that he might have swallowed a tooth. A few days later he returned and said that the slight cough had continued until about two days afterwards when he coughed up a tooth, which, judging from the slight symptoms, perhaps had been lodged well above the vocal cords. On the other hand, it is more probable that it may have passed down the trachea and become fixed in a bronchus.

*Case of Respiratory Obstruction in a Patient, in whom part of the Tongue and a large Mass of Glands had been excised.*—Patient, a thick-set oldish man, had had the greater part of his tongue, part of the floor of the mouth, and a glandular mass excised for cancer. The parts about the base of the tongue were practically fixed, and he could not open his mouth very widely or project his tongue forward. He needed extraction of several teeth. So much of his tongue had been removed, and it was so fixed, that I did not think there was much chance of respiratory obstruction. Local anæsthesia was impossible because the mouth could not be sufficiently opened. Gas was administered. He went under quietly, a lower tooth was extracted, but he quickly came round, so I had to begin again. When the operator was extracting the upper roots the head was pushed back a little and respiration became obstructed; gas was stopped. He had brisk reflexes and small pupils. Extractions were quickly completed in a few seconds, then respiration practically stopped. I tried to pull his tongue forward, but the tip only came level with the teeth. I could not hook it forward nor could I reach the epiglottis with my finger. Then his reflexes became dull, his colour blue, he made attempts to breathe but got practically no air in; so he was placed prone with the head hanging over the edge of the head-piece, and Schäfer's method of respiration was

tried. After several minutes (it seemed much longer) I got out my instruments and prepared to do laryngotomy when the patient was apparently *in extremis*. Fortunately, however, he managed to get in a little air and gradually recovered.

This case occurred a good many years ago. Nowadays one would have passed a mouth-tube and probably respiration would have been quickly re-established. It is the only time I have taken out my tracheotomy case in dental work.

*Case of Swallowed Sponge.*—Patient, a woman, aged about 50, very nervous, stupid and alcoholic, who would not do anything she was asked to do. She took an enormous amount of gas nasally to go under, and came round in a few seconds, so I gave ether, after packing a sponge in the mouth, and got her under with difficulty. Two teeth were extracted, then round she came, and developed blueness and much spasm of the jaws. I was using several sponges in the inhaler and believed that she swallowed one. Spasm, blueness and cough continued, and the patient complained of pain in her throat. After about five minutes she felt something in her neck, and had difficulty on swallowing. She recovered, swallowed some tea, and appeared little the worse, and now felt nothing in her neck. On arriving home she felt pain and discomfort in the middle of the front of her chest. Then she took an aperient, and about 2 a.m. the bowels acted and the pain ceased. She did not look for the sponge. A wet slimy sponge which had been wrung out of lysol would have easily been swallowed. If so, it might soon have been passed or probably would have rapidly disintegrated. The moral to be drawn is to tie sponges together, and to use fairly large sponges.

The following case is of surgical interest:—

*Tremendous Hæmorrhage after Extraction of One Tooth: Cirroid Aneurysm.*—Patient, a girl, aged 9. After the extraction of a first upper molar tremendous bleeding occurred, the blood filled her mouth and drenched her clothes. She rapidly collapsed. For a time I managed to control the bleeding, which I thought was from an abnormal artery, but soon had to send for a surgeon, who succeeded by means of pressure over a large pad in stopping the hæmorrhage, which did not recur. About fifteen months later "the second upper molar was found to be loose as though it were placed in a rubber sack which was resting on water." Soon afterwards there was bleeding in the night from the region of this tooth, and next day more bleeding. The doctor found a soft, spongy, pulsating mass to which two teeth were attached. The surgeon, Mr. Heaton, visited the patient and found her blanched from hæmorrhage. Pressure was applied, the carotid artery was compressed, the second upper molar was extracted, and furious hæmorrhage followed, arterial in character, and so prolonged that the external carotid artery was tied. The second upper bicuspid was extracted, and then the bleeding became as furious as ever. Therefore the common carotid was tied, but after so doing hæmorrhage persisted, and could only be controlled with the greatest difficulty. Pressure had to be maintained for eight or ten days. After the teeth had been extracted there remained a cavity as big as a walnut. The diagnosis appeared to be that of a plexiform angioma or cirroid aneurysm.

One patient, who was very excited during induction, and after anæsthesia, developed jaundice for a few days. He had twice before been yellow for four or five days after taking nitrous oxide.

#### *Two Cases of Pneumonia after Anæsthesia.*

*Case I.*—Patient, a man, aged about 50, in very bad condition. Ether was given for the extraction of many very septic teeth, and for a small rectal operation. He died in a few days of septic pneumonia.

*Case II.*—Patient, a young woman, who had ether only, in the recumbent position, for the extraction of twelve teeth. She was subject to a "little cough." A rigor occurred next day with pain in the left side, and pneumonia developed, from which she recovered.

#### SUMMARY AND CONCLUSIONS.

(1) Dental operations should be concluded at one sitting if reasonably possible, even if it means resorting to a major anæsthetic. The patient will prefer it.

(2) Nitrous oxide, by the prolonged method, is the best routine anæsthetic for qualified men and students. For difficult cases, deep ether, followed by light chloroform anæsthesia, is best.

(3) Major cases should be dealt with in hospital.

(4) Ether, with or without a little ethyl chloride, from an Ormsby inhaler gives the best results in the case of small children and troublesome adults.

(5) Shock after long dental operations under gas develops mostly as the result of unsatisfactory anæsthesia and asphyxia, and the addition of a little ether, or an etherized sponge greatly improves the condition of the patient.

#### DISCUSSION.

Mr. WARWICK JAMES showed an instrument which he had devised when working upon the fractured mandible in connexion with war wounds. He said that he found it of value in all cases of anæsthesia, whether the operation was upon the mouth or not, as by its use the mandible could be advanced and the restriction to respiration removed. He stated that during the ten years he was Dental Surgeon to the Hospital for Sick Children, Great Ormond Street, ethyl chloride was almost entirely used, as it was found to be the best anæsthetic for quite young patients, particularly in dealing with a large number of teeth. He held that there should be no difficulty in dental anæsthesia, short or long, if the dentist kept the mandible forward.

Mr. CARTER BRAINE stated that in the case of multiple extractions he preferred to administer nitrous oxide followed by ether with the patient seated in the dental chair, provided the extractions did not take more than half an hour to complete, using the Ormsby inhaler. He considered that the dental surgeon was awakening to the best mechanical advantage derived from this position, particularly when extracting lower molars. When it was necessary to re-apply the inhaler he bent the patient well forward so that all blood could fall out of the mouth into the inhaler, and none be swallowed or inhaled. This could not be done with a Clover's inhaler, as the airway would become blocked with blood-clot. One application of the ether inhaler was sufficient in the bulk of the cases, but if re-application was necessary then this should be done before the patient had recovered sufficiently to be conscious of it. When the operation was likely to last for a longer period, say one hour or more, then he preferred the patient to be in the horizontal position on the operating table, and after anæsthesia had been induced with nitrous oxide and ether, or chloroform and ether, he continued with chloroform given through the nasal catheter, and it was surprising how light an anæsthesia was then requisite. The chief difficulty in this position was the keeping the head fixed in the requisite position; the ordinary pillow was quite useless, but a sandbag made in the shape of a large horseshoe had been found most serviceable. This should be placed with its convexity under the cervical region, pointing towards the trunk, with the prolongations of the horseshoe extending along either side of the head towards the occiput; this rendered the head comparatively firm during the extractions. The employment of light gauze packing at the back of the mouth in all cases was strongly advocated, in order to prevent all the stumps, &c., falling back into the pharynx.

Mr. BELLAMY GARDNER expressed his surprise that the benefits of nitrous oxide and oxygen were not more generally recognized for dental anæsthesia, the greater control of the asphyxial factors during the operation being so marked. He thought that when a patient was sent to a nursing home the operation should take place in the

sitting position, upright; a dental chair being hired for the purpose if necessary, so that the dentist should, as far as possible, be allowed the same conditions to which he was accustomed in his own surgery. There was no objection to the administration of ether in the sitting position if the anæsthetic were administered throughout in that posture from the beginning. There was danger in raising patients upright who had been first anæsthetized lying flat.

Mr. H. E. G. BOYLE said he was a little surprised to find that Dr. McCardie used gas alone for dental extractions, and thought that better results were obtained by giving gas and oxygen; for he thought that the addition of the oxygen helped to overcome the feeling of suffocation that was sometimes complained of by people to whom gas alone had been given. His own practice was to obtain anæsthesia with gas and oxygen, and to maintain it with gas through a nose-piece, and in this way he produced as long an anæsthesia as the dentist needed. For major dental work he preferred to have the patient in a nursing home, giving a preliminary hypodermic of either morphia and atropine or morphia, atropine and scopolamine according to the nature of the case and type of patient, then to give the gas-oxygen-ether or the gas-oxygen-C.E. combination, continuing the anæsthesia by means of nasal tubes, and having some form of suction pump at hand to remove the blood, and minimize sponging. He did not care very much for ethyl chloride, and thought that for difficult children open ether was probably the best anæsthetic to give. On the point that had been raised about the physician selecting the anæsthetic, he thought that the selection should be left entirely to the anæsthetist to give what he felt was best and safest for the patient.

Dr. F. E. SHIPWAY was surprised to hear that Dr. McCardie advocated giving stimulants before anæsthesia with gas: he had always been under the impression that they excited the patient and made anæsthesia more difficult. Probably the explanation was that he had not given them in such large doses as Dr. McCardie, who must have succeeded in producing a narcotic effect. With regard to anæsthesia for major dental operations he (Dr. Shipway) advocated ether, and preferably intratracheal ether if the operation was on the lower jaw and likely to be prolonged and difficult; the difficulties of anæsthesia during the extraction of a lower buried wisdom tooth could not be solved satisfactorily in any other way. He agreed with Mr. Boyle that some form of suction apparatus was helpful in removing blood and mucus, but it did not prevent obstruction of the breathing caused by the presence of sponges and instruments in the mouth, depression of the mandible and the pushing back of the tongue.

Mr. FRANK COLEMAN said that the extra time allowed by the nasal method of administration of nitrous oxide was of great benefit to the dentist, as the tooth could be taken out more carefully and with less damage to the jaws and consequently less after-pain. Previous to the introduction of this method, the dentist had usually to work under a time limit anæsthesia of half a minute or so following the removal of the mask and in those days it was generally regarded as being a dexterous feat to remove the four first permanent molars under a single administration of nitrous oxide. As a matter of fact this was usually far from a satisfactory or skilful procedure, as time being so brief it only allowed for each tooth to be wrenched forcibly once, or at the most twice, inwards and outwards, with much damage to the alveolus and uncertainty in the result. An operator of experience developed a very acute sense of touch and could detect from the feel of resistance in the tooth how much force he could safely employ, and when, and when not, he could expedite the operation. During the war and in the forward areas where nitrous oxide was unattainable, he (Mr. Coleman) had found that "open ether" preceded by an injection of morphine and atropine an hour and a half or two hours previous to the administration gave the best results for dental cases, apart from those that could be treated satisfactorily under local anæsthesia.

Dr. CECIL HUGHES agreed that for most routine cases gas and air gave excellent results, especially by the nasal method, which patients found far pleasanter than the oral method. He did not agree with a previous speaker that gas and air produced a more suffocating induction than gas and oxygen. Provided that the patient could be persuaded to breathe slowly and quietly, and that the gas was not administered under pressure, the production of a sensation of suffocation could always be avoided.

## 22      McCardie: *General Anæsthesia in Dental Surgery*

Mr. I. W. MAGILL asked Dr. McCardie whether in those cases requiring a long anæsthetic he had ever used intratracheal insufflation through the nose. With a second catheter passed through the free nostril to provide for expiration, he (Mr. Magill) thought that this method gave the nearest approach to a physiological anæsthetic. The mouth could be plugged as desired and the operator given ample time and a clear field. Combined with a modern suction apparatus the method seemed to him almost ideal.

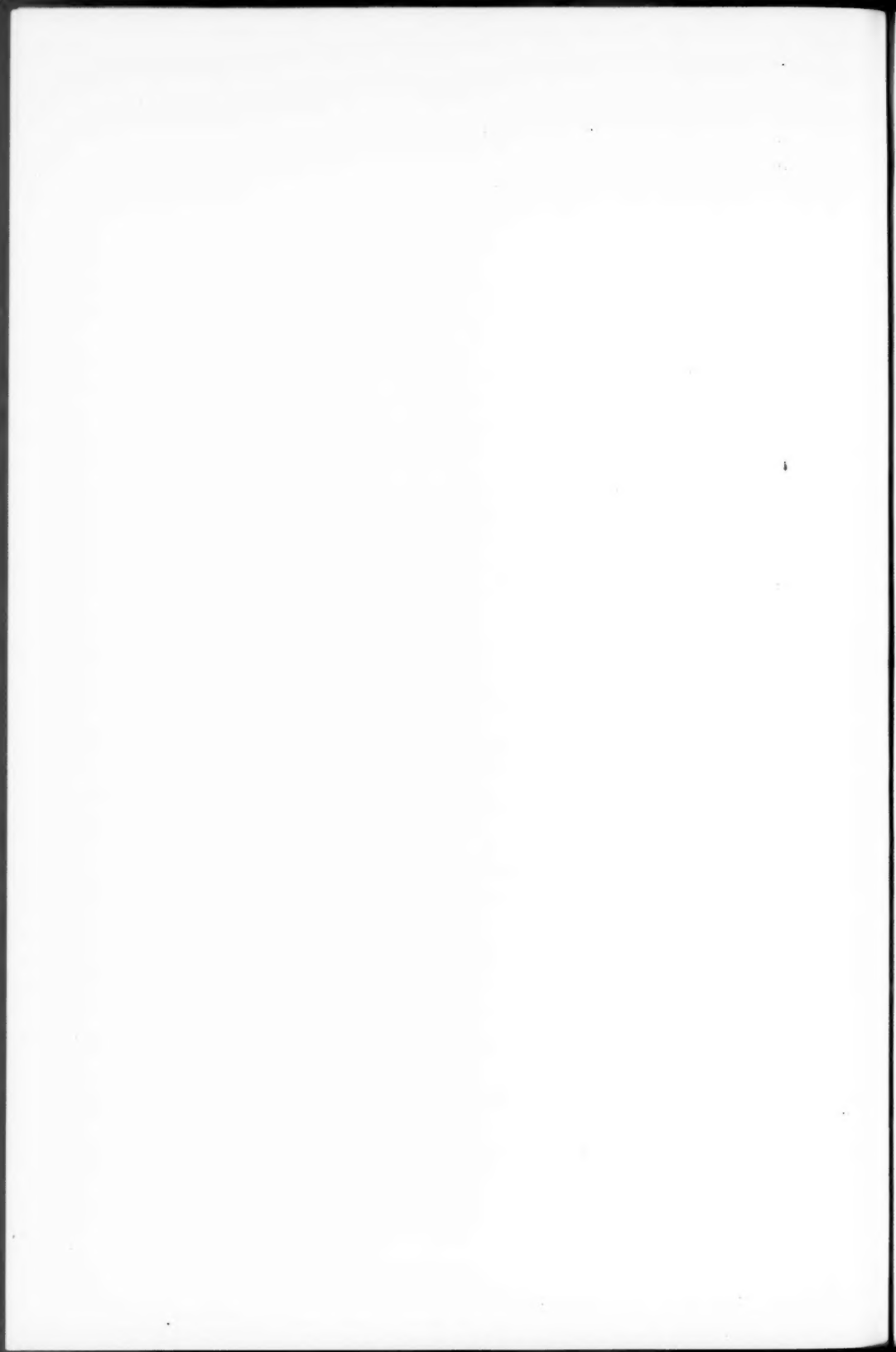
Mr. F. ST. J. STEADMAN emphasized the great importance of patients having a meal an hour or two before the administration of nitrous oxide for the extraction of teeth. He had frequently observed that patients who had had teeth removed under nitrous oxide at about 10 a.m., after having had no breakfast, felt faint after the operation, and that they were upset for the remainder of the day, whereas patients who had had their usual meal showed no such after-effects as a rule. It was unfortunately a very common practice among general medical practitioners to advise patients to go without their breakfasts before the administration of the anæsthetic. He was absolutely convinced that this advice was wrong.

Dr. HUGH PHILLIPS said that at the Hospital for Sick Children, Great Ormond Street, practically nothing but chloride of ethyl was used in the dental department, and was quite satisfactory. He found that London dental surgeons were altogether opposed to general anæsthetics being administered in their own consulting rooms owing to the length of time before the patients were fit to go home and also on account of the mess caused by the administration. He also supported the use of alcohol taken beforehand for those accustomed to it.

Mr. C. J. LOOSELY agreed that patients who took gas badly took ether well, i.e., those who tended to become rigid, or struggle and phonate under gas, were quite placid on addition of ether and were never sick nor upset after it. He thought that the dropping of ether or chloroform upon the sponge in the patient's mouth was a somewhat crude practice. He had had a modified Clover attachment made to be placed between the bag and the nasal tubes, so that in these special cases a small amount of ether could be added to the gas. He had found that this answered admirably, these patients recovering from the ether as quickly and as cleanly as the average patient did after gas alone. Dr. McCardie did not mention the depth of the gas anæsthesia he advocated. He (Mr. Loosely) attached much importance to this in nasal work, and found that by increasing the depth too much a good nasal breathing would be changed to oral breathing, probably owing to the physical need of the patient for more oxygen. He did not care for the Ormsby inhaler owing to the difficulty of keeping the bag full of air. The irregular breathing of a patient, if nervous, might cause the patient to take an inspiration when the anæsthetist expected him to make an expiration, thus emptying the bag and getting the full dose of the anæsthetic in on breathing.

Dr. MCCARDIE (in reply) said that he believed that it was better for students to be taught to administer nitrous oxide alone because the signs of anæsthesia were so definite; it was used in practice by both doctor and dentist, and the necessary apparatus was simple. By properly regulated admission of air nearly as good a result could be attained as by the addition of oxygen. One or two speakers advocated ethyl chloride alone for children. In his opinion ethyl chloride was to gas what chloroform was to ether. A little ether added to ethyl chloride safeguarded it, prevented shock, and lessened after-effect. In a long case Mr. Carter Braine preferred ether by the Ormsby inhaler and readministration when necessary. Readministration, however, in a patient who was partly round and whose mouth held more or less blood, was not so good as continued anæsthesia prolonged by a little chloroform or a mixture of chloroform and ether. There was no doubt that the Ormsby inhaler had great advantages over the Clover for dental work. The sitting-up position was certainly the best one for most difficult cases, which could be operated on in the dentist's rooms, but it was perfectly easy when the patient was recumbent to raise his head nearly to a right angle with the body, for extraction of lower teeth. With regard to the question of concluding the operation at one sitting, it was for the dentist to decide as to the toxic factor and how much was to be done at one sitting. It was for the anæsthetist to

decide as to the patient's ability to undergo the complete operation. Of course the kind and degree of sepsis varied a great deal. In the milder form the whole mouth might safely be cleared without fear of after-danger. In the severer toxic form less work must be done at a time. It had been suggested that with continuous gas the students wasted time over extractions, but it seemed to him that by taking more time they did less damage and could be better taught how to extract. Continuous gas gave a much better chance to both operator and patient. In the provinces they could comparatively seldom take dental patients to a nursing home or operate in their own homes, so that they had usually to be prepared to do what was necessary in the consulting room or the dental hospital. From what had been said it seemed that in London private patients could better afford the time and money for admission to a nursing home than was the case in his (Dr. McCardie's) district. Why did patients take gas badly? He suggested as possible causes, nasal obstruction, psychic resistance, fear, alcohol and other drugs, lymphatism, or a low barometer. He had not known addition of oxygen to cause distress and asthma, but he thought that there was more sickness afterwards. It was certainly advisable in cases of high blood-pressure. He preferred not to restrict his patients by bands or mechanical contrivances. He was glad to find that brandy was approved as a stimulant and narcotic. It had been said that alcoholics needed more oxygen than other patients did. Probably this was so; they certainly needed more brandy and more ether. With regard to Mr. Boyle's suggestion that there was less feeling of suffocation when oxygen was added to gas, he believed that the feeling of suffocation was largely a matter of too widely open a mouth, especially when a centre gag was used and there was undue pressure of gas or obstruction in the nose. During induction quiet breathing must be insisted upon. Mr. Boyle, after beginning with gas and oxygen, continued with anæsthesia with gas and air. He (Dr. McCardie) would have thought that there would be advantage in reversing the process, that is, adding oxygen when obstruction was caused by the dental procedure. With regard to shock, he thought that the extraction of the four six-year-old molars in children was the most severe operation, especially if the extractions were difficult, and he was inclined to believe that ether should be preferred to nitrous oxide. Clearing the mouth might or might not be a major operation. It was for the dentist to decide what was a major operation. Mr. Steadman advocated a good meal two hours before gas anæsthesia. He (Dr. McCardie) did not agree with this recommendation. It was no doubt unpleasant to have a major anæsthetic administered in the dental surgery, but if these difficult cases were dealt with at the end of a morning, or better, at the end of the day, when the fine work needing steadiness and delicate touch was over, there could be little objection. During administration he tried to modify the depth of anæsthesia as occasion required. Success depended on the ability to vary the doses of gas. He generally endeavoured to keep very slight blueness and regular respiration, using a deeper anæsthesia for extraction of upper teeth. The anæsthetist must be a "quick change" artist. Years ago he interposed a Clover inhaler between the bag and the nose-piece and had very good results, being able to add at will small doses of ether to the gas. He had never used intratracheal etherization in dental work.





## Section of Anæsthetics.

President — Dr. A. L. FLEMMING.

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### On Systematic Examination of the Heart.

By J. STRICKLAND GOODALL, M.B.

IN accepting the invitation to read a paper here this evening, I was not a little influenced by a sympathy that I have always had for the anæsthetists since those far off days when I myself was anæsthetist to a small special hospital. For certainly, in those days at any rate, it seemed to me that the anæsthetist took a good deal of the responsibility, submitted to a good deal of personal discomfort and received in return a good deal of blame if things "went wrong" and very little credit if things went well. More recently it has struck me as a spectator, that the patients themselves often attach very slight importance to the anæsthetic and its administration, for whereas they regard it as of vital importance to secure a first-class surgeon to perform a simple operation, they frequently appear to think that "anyone" can give the "stuff." Another reflection that has frequently been uppermost in my mind is the lack of real co-operation between surgeon, physician and anæsthetist, and, still more often, the very casual way in which a physician will, if consulted in the matter, express an opinion that a case can stand "any operation" without trouble being taken to ascertain the nature and duration of the operation it is proposed to perform. As one who has for many years past examined a large number of goitre cases preceding operation, this fact has been specially brought home to me.

The main object of all concerned in any operation, whether it be the physician, surgeon or anæsthetist is, I take it, to reduce the risk, danger and discomfort of the patient to a minimum, and this ideal is, I think, most likely to be achieved by: (1) a thorough and complete examination of the patient, and especially of his cardiovascular system; and (2) a more complete co-operation between physician, surgeon and anæsthetist.

It is with the first of these, viz., the complete investigation of the cardiovascular system, that I propose to deal to-night, and here I would say that I look forward to seeing at no very distant date, a properly equipped cardiological department, controlled by a properly qualified cardiologist, in every general hospital where operations are performed, and in which a *systematic routine* examination of *all* cases will be carried out in advance of any operation, and without which no operation, other than those of the most urgent and acute type, will be performed. I fully realize that, in many cases, the patient is so desperately ill, and the operation one of such urgency, that any examination but the most superficial is impossible, but I still maintain that

the more systematic and thorough that examination is, the more judicious will be the choice of the anaesthetic and the less the risk to the patient. The patient should be examined under three separate conditions, viz., lying, and, if possible, standing, and after exercise.

To the trained cardiologist, much may be obvious. The well-developed, overgrown, robust appearance of the young aortic, with his pale lips, throbbing neck, and over-development of muscles, &c.; the small undergrown red or reddish-blue "congenital mitral stenosis;" the "pinched" patchy face, with the tortuous temporal arteries, and the often wasted body typical of cardio-vascular degeneration; the yellowish anxious face of infective endocarditis; the large white face of renal disease; the blue face of congenital or failing mitral disease; the worried appearance of the patient with angina, or the distressed pale face of pericarditis, can often be recognized at a glance, or at any rate the underlying condition can be very shrewdly guessed.

The respiratory rate, depth and rhythm, cyanosis, engorgement, pulsation or otherwise of the jugulars, presence or absence of carotid pulsation, thyroid enlargement, clubbing of the fingers and, if present, whether blue (congenital heart disease) or white (infective endocarditis), swelling of the ankles, &c., can, in fact, all be noted before the patient has reached the examination chair.

Should the patient be confined to bed, particular attention should be directed to the position in which he lies or which he assumes. When the chest has been exposed, its shape and movements, any bulging of the precordium (cardiac disease in early life before the chest has ceased growing) should be noted, also the apex beat, position and character, special attention being directed towards whether it is heaving (the true sign of cardiac hypertrophy) or slapping in character. And here I would remind you that a slapping apex beat means a poorly contracting ventricle, and that this occurs under three sets of conditions, viz.: (1) When the muscle is poisoned (toxic myocarditis); (2) when the muscle has degenerated (myocarditis or cardio-vascular degeneration); (3) when the ventricle is badly filled, badly stretched, and so contracts badly, e.g., mitral stenosis. Systolic recession, indicative of adherent pericardium, should be looked for, not only in the region of the apex, but in the region of the epigastrium and also in the back (Broadbent's sign).

The rate should be carefully noted and compared with that of the pulse. If any irregularity is present, frequently the nature of the irregularity can be made out with the greatest ease. It is very often possible to diagnose auricular fibrillation, &c., by palpation. The presence or absence of thrills should be noted. If present they should be timed with the carotid, their exact position noted; it should also be noted whether they are constant or intermittent in character.

The commonest thrills of all are of course: (1) The apical presystolic thrill of mitral stenosis, which may be either constantly present as it is during the stage of full "compensation," when the auricle is well hypertrophied and its contractility unimpaired or intermittent, being possibly present only when the patient is lying or after exercise. Usually the presystolic thrill is associated with a regular rhythm, but occasionally it exists with an irregular rhythm due to either auricular fibrillation or auricular extrasystoles. Roughly speaking, an intermittent presystolic thrill, with a regular rhythm occurs very early in mitral stenosis, before the auricle is fully hypertrophied; intermittent presystolic thrills with an irregular rhythm are found at the commencement of failure. Cases of this type can certainly be well treated and the patient's condition materially improved before an anaesthetic is given. (2) A systolic

thrill at the apex (mitral regurgitation), pulmonary base (pulmonary stenosis), aortic base (aortic stenosis or aneurysm) or a pericardial friction may be present. Any abnormal pulsation should of course be noted and investigated.

The abdomen should be examined for distension, and the importance of ascertaining distension of the abdomen, with its very serious cardiac embarrassments, should not be underestimated. The presence of engorged veins, absence of respiratory movement (? pericarditis) and the presence of ascites should all be observed. Special attention should be directed towards the liver, while the spleen should also be palpated. As regards the arms and the hands, the condition of the brachials is of tantamount importance. Note whether they are visible, thickened; if the latter, note whether the thickening is uniform or otherwise, bearing in mind that if moniliform in character, this usually indicates involvement of the muscle coat as well as of the intima.

The locomotor artery always signifies two conditions, viz., a rigid vessel and a hypertrophied heart.

At this stage, one may well investigate the pulse: (a) Rate: whether abnormally fast, or abnormally slow; a pulse-rate below 40 means either heart-block or physiological bradycardia; the latter occurs in well trained athletes or after certain debilitating toxic conditions. Which of the two conditions is present can be ascertained by exercise, if the patient is well enough. The heart-block pulse accelerates little, if at all, whereas the athlete's pulse doubles its rate on exertion and does not "climb" like a normal pulse. Or the electro-cardiograph or some other instrumental method can be employed.

(b) Rhythm: Whether regular or irregular; if the latter the nature of the irregularity should be investigated. For the sake of convenience we may divide them into (1) regularly irregular or (2) irregularly irregular.

The most common regular irregularities are: (1) Pulsus bigeminus or pulsus trigeminus, indicative of myocardial irritability; (2) sinus arrhythmia (where the pulse speeds up during inspiration, and slows down during expiration), indicative of (according to some but not the writer) a healthy myocardium; and (3) pulsus alternans, indicative of failing contractility. This is a sign of very grave prognostic significance.

The commonest irregular irregularities are: (1) The perpetual irregular pulse due to auricular fibrillation. Here the beats not only follow one another at irregular intervals, but are of unequal strength and volume. In addition the pulse-rate differs from the apex-rate, while the irregularity is increased by exercise. This condition may be due either to mitral stenosis, myocardial degeneration or myocardial toxæmia (Graves' disease). (2) The irregularity due to irregularly occurring extrasystoles. This is indicative of myocardial hyperirritability, which in turn may be the result of either fatigue, inflammation, degeneration, or toxæmia. (3) The force estimated by the impact against the finger depends upon the condition of the myocardium, e.g., in aortic regurgitation, where the left ventricle is hypertrophied, the force is considerable, whereas in mitral stenosis, where the left ventricle exercises but little force, the impact is very slight indeed. (4) The volume estimated by the lift and the duration of the wave equals the output of the heart. While (5) the tension is estimated by obliteration force and the blood-pressure.

On auscultation attention is first directed to the heart sounds; at the apex the first sound in a healthy heart is long, dull and booming, and is due to two factors, namely, the contraction of the ventricular muscle and the vibration set up in the auriculo-ventricular valves and chordæ tendineæ. Any interference

with the contractility of the muscle, or the closure of the auriculo-ventricular valves will necessarily modify the character of the first sound, which may be (1) shortened, indicating a poorly contracting ventricle, due to either inflammation, degeneration, toxæmia, or non-stretching (e.g., mitral stenosis); (2) reduplicated, due either to the non-synchronous contraction or closure of the auriculo-ventricular valves; (3) weak or suppressed, due to pericardial effusion, emphysema, myocardial degeneration, &c.; or (4) modified or replaced by a murmur or adventitious sound.

Likewise the second sound at the apex, due to the closure of the aortic and pulmonary semilunar valves may be (1) distinct; (2) reduplicated, when the normal relationship between the pulmonary and systemic tensions are altered, or when the aortic and pulmonary valves do not close simultaneously; or (3) modified by the presence of a murmur as in mitral stenosis.

*At the base* the aortic second sound normally short, sharp and flapping, and due to the closure of the aortic semilunar valves, may be (1) accentuated—a high peripheral resistance and a high blood-pressure; (2) ringing, indicative of atheroma of the aorta usually with dilatation and rigidity of the valves. This condition, most characteristic to those who are familiar with it, is completely different from an accentuation and has a totally different significance. It may, or may not be, associated with a high blood-pressure. (3) Absence of the aortic second sound means either that the aortic valves do not close owing to injury or destruction, or that they close so quietly that they do not produce an audible sound. This latter condition sometimes occurs in a more or less acute aortitis; (4) modified by the presence of a murmur which replaces it partially or entirely. And here one might point out that diastolic murmurs produced at the aortic valve must be listened for in three different positions: (1) Over the lower part of the sternum. This murmur is usually to and fro in character and is found typically in those cases of aortic regurgitation in which the whole valve is more or less damaged. (2) Over the junction of the second right costal cartilage with the sternum. Here the murmur is harsh, loud, and just beneath the stethoscope. This is particularly associated with damage to the anterior cusp which is the lesion most frequently liable to be connected with pains in the chest (angina). (3) Over the pulmonary base (third left costal junction). Here the murmur is soft, blowing but distant, never harsh in character. It is indicative of damage to the left posterior cusp which is always, in the writer's experience, secondary to a mitral lesion. This murmur should be carefully listened for in any case of mitral stenosis, where the face is pale, where the left ventricle is large, where the pulse is medium to large in volume, or where the blood-pressure is high.

*The pulmonary second sound*, due to closure of the pulmonary valves, is also short, sharp and sudden, and in young adults less distinct than the aortic second sound, but generally the reverse in young children. It in turn may be accentuated (high pulmonary tension) as occurs in mitral stenosis and various lung conditions, reduplicated, as also occurs in mitral stenosis and other pulmonary defects, or modified by a murmur.

Auscultation thus provides one with accurate information regarding the state of the myocardium and the functioning of the different valves. In a healthy heart the intensity of the first sound at the apex bears a definite relationship to the intensity of the aortic second sound at the base. This relationship can be easily ascertained by means of a differential stethoscope. In a healthy heart the ratio is 2—1. Should the myocardium be degenerated,

there is a ratio approximately of 1—1. The differential stethoscope is thus an additional means of investigating and assessing the condition of the myocardium.

The significance of the different murmurs is too well known to need description, but graphic photographic records of them can easily be obtained, and their character fully analysed by the means of the cardio-phonograph.

A special type of murmur, the atonicity murmur, is perhaps worthy of brief mention. This murmur, which may be heard over either apex or base, is soft and blowing in character and very variable. It may be more or less constantly present, standing, lying and after exercise. If present at the apex, it may be local or conducted outwards to the axilla. It may be absent at rest and only produced by exercise. On the other hand, it may be present at rest and disappear after exercise. This atonicity murmur, so called, indicates lack of tone in those rings of muscle which support the mitral and other valves, and it is the degree of atonicity present which determines the presence or absence of this murmur.

As above mentioned, the case must be examined if possible after exercise, so that one might well here consider the more important effects of exercise on the heart.

(a) *The Rate.*—The normal heart responds to exercise by a gradual increase in rate. The increase is more or less uniform, the heart climbing up as exercise is increased. The normal heart rarely speeds up to over 150. It rapidly returns to normal on ceasing the exercise. A soft atonic or poisoned heart responds to exercise by undue acceleration, and very slowly settles down to its normal rate; while, in certain diseased conditions of the heart one gets impaired acceleration; the rate practically speaking showing no alteration at all. This may occur in very fast hearts (e.g., auricular flutter), or in very slow hearts (e.g., heart-block). Lastly, in this connexion one might point out that in a well-trained physiological heart, such as one meets with in young, highly-trained athletes, the rate does not climb on exercise, but suddenly doubles (e.g., at the commencement of the exercise, the rate may be 42, and on exercise suddenly becomes 84)—the so-called athletes' reaction.

(b) *Rhythm.*—On exercise the rhythm of the heart may be profoundly modified. (1) An irregularity may be produced, and here I would emphasize the fact that any heart that becomes irregular on exercise is a damaged heart. The most common irregularities which are brought out by exercise are extrasystoles (indicative of myocardial fatigue), auricular fibrillation (indicative of inco-ordinate auricular contractions secondary to degeneration, toxæmia, &c.), alternating (indicative of failing contractility) a sign of very grave significance. (2) An existing irregularity may be abolished. Practically speaking, the only irregularity which is abolished by exercise is the irregularity due to extrasystoles, *when they are not due to fatigue*. (3) An existing irregularity may be increased. This is true of auricular fibrillation or fatigue extrasystoles.

(c) *Sounds.*—Under the influence of exercise, normal heart sounds may be reduplicated or modified by the production of adventitious sounds or murmurs. In early mitral stenosis, and in soft hearts, atonic murmurs may either appear or disappear according to the underlying condition of the myocardium.

(d) *Thrills.*—Thrills may be actually produced. This occurs in early mitral stenosis, when increased filling of the auricle results in increased stretching, and increased contraction, and so produces a thrill, presystolic of course in time. An existing thrill may be increased under similar conditions or abolished by exhaustion.

In all systematic routine examinations of the heart, electro-cardiographic records should be obtained, as these provide one with invaluable information as to the condition of the myocardium. The contraction of the heart muscle is accompanied by, or slightly preceded by, an electrical variation which can easily be photographed and estimated. Each part of the photographic curve (electro-cardiogram) thus obtained corresponds to activity in a certain part of the heart muscle, and by careful investigation of each part of the curve accurate information can be obtained, not only of the underlying state of the muscle, but also of the exact way it is functioning at the present time.

X-ray examination of the heart enables one to tell the form, position and size of the heart; to analyse the beat, estimate its tone and rhythm, and also enables one to judge the condition of the pericardium; the state of the great vessels, and of the mediastinum generally.

Sir BERNARD SPILSBURY described and analysed the Heart Conditions found Post-mortem among Fatalities connected with Anæsthesia.

### The Action of Chloroform on the Heart.

By A. G. LEVY, M.D.

So far as I can gather from leading text-books there is a consensus of opinion that, provided the circulation is not seriously affected, inhalation anæsthesia is generally regarded as a safe procedure in cases with cardiac disease. As a matter of fact individuals affected with valvular disease are frequently "good subjects," especially for chloroform anæsthesia. Yet, if a fatality should occur, there is a tendency to lay the responsibility on the heart, rather than on the anæsthetic.

Recent knowledge of the mechanism of chloroform syncope must lead us to recast our notions of the relation of cardiac activity to this disaster. So long as chloroform was regarded only as a cardiac depressant, and every case of syncope as a manifestation of overdosage, then it was perhaps permissible to draw the inference that cardiac disease and chloroform syncope were inter-related. It happens, however, that the condition underlying chloroform syncope, viz., fibrillation of the ventricles, is essentially a manifestation of a heart which is healthy except in so far as it is affected by chloroform, and, in fact, a heart with depressed muscular action is less subject to this form of syncope; so also is ventricular fibrillation less likely to occur in the presence of a low blood-pressure. I do not say that there may not be body conditions predisposing to fibrillation in some individuals; that is a matter for speculation, but I do say that there is no reliable evidence that endocardial lesions or muscular degenerations, fatty or otherwise, predispose in any way to chloroform syncope.

There is no doubt that a fatty degeneration of the heart muscle in some degree is found in many cases of death under chloroform, but fatty degeneration is found in such numerous pathological conditions (indeed, it is readily brought about by the inhalation of chloroform itself) that I greatly doubt whether any inference of causal relationship is warrantable.



Let me now pass to those cases of chloroform syncope in which an enlarged or persistent thymus is found post mortem. I do not propose to discuss the conditions of *status lymphaticus* at length, as I do not think there is sufficient satisfactory material available, but I can say this—it would appear that in those cases in which pure chloroform was employed, death followed either a brief and light administration, as for the operation for tonsils or adenoids, or some irregularity of administration which I have described as conducive to ventricular fibrillation. Hence, I believe that these fatalities are essentially cases of chloroform syncope and, as such, are avoidable.

There is, however, one cardiac disorder which requires a word of caution—that is heart-block. Deep chloroform anæsthesia frequently leads to heart-block in animals, generally taking the form of a complete dissociation of auricular and ventricular beats. I do not think that this in itself is harmful, but it is conceivable that pre-existing heart-block may be accentuated by chloroform, and I should therefore consider it undesirable to employ chloroform in such cases. About ether in this connexion I cannot speak, but perhaps some members of this Section may be able to supply clinical information on this subject.

It has been suggested that when the heart is affected by ventricular extrasystoles, they are an evidence of cardiac irritability and that therefore chloroform is contra-indicated. I do not think that this is necessarily the case; extrasystoles have distinct sources of origin, and it does not follow that chloroform will accentuate those arising in a different way; thus extrasystoles arising from a raised blood-pressure are certainly not exaggerated by chloroform, and I am inclined to think that the danger from pre-existing extrasystoles is negligible; but, here, again, clinical information is desirable. A predisposition to ventricular fibrillation has been presumed in exophthalmic goitre, and the instances of sudden syncope in persons suffering from this condition when not under an anæsthetic have been ascribed to this cause. For this reason chloroform would appear to be undesirable, but, in addition, the light anæsthesia which is required for technical reasons in operations in such cases of course rules chloroform entirely out of the question.

#### DISCUSSION.

Dr. A. L. FLEMMING (President) said that the members of the Section were indebted to Dr. Strickland Goodall for much useful guidance in connexion with some of the most anxious cases that they were, at times, called upon to deal with; Dr. Goodall's remarks would inspire confidence where functional disorders were concerned, and would impress on them the real danger which might be incurred by the use of chloroform in the presence of myocardial degeneration; his description of the variable operation risks in the three stages of Graves' disease, as indicated by the behaviour of the blood-pressure, would be especially welcomed. With reference to bradycardia, he (the President) had occasionally met with instances in which deep anæsthesia had given rise to an ominous slowing of the pulse-rate. There was one detail of practice in which a certain laxity seemed lately to have arisen; after examining patients to determine their fitness for operation physicians had advised them personally as to the type of anæsthetic most suitable in their respective cases, and the anæsthetists concerned had been called upon merely to dispense the prescriptions thus produced, without having been consulted in the selection of drugs; the result of thus tying the anæsthetist's hands seemed, to him (the President) to be unwise.

Dr. JOHN PARKINSON appreciated the need for a more extended examination in any patient whose heart was in question before he was submitted to an anæsthetic. The value of simple palpation with the whole hand in the region of the apex-beat was



great, as Dr. Strickland Goodall had insisted. He could not agree, and it was not the current opinion, that extrasystoles, when they were the only sign suggesting anything amiss, could be looked upon as of serious significance. Their widespread occurrence among healthy men was generally recognized. The essential fact to establish was the presence or absence of more conclusive signs, not forgetting an inquiry into breathlessness and other symptoms. For immediate use when there was circulatory failure during an anæsthetic they had no efficient medicinal remedy, and this fact had doubtless led to greater inquiry into the possibility of massage and mechanical stimulation of the heart. It was not sufficiently known that patients in whom auricular fibrillation was present, with a frequent irregular pulse, could be raised to a higher standard of myocardial efficiency by a week's course of digitalis in preparation for an operation. This not only reduced the risks at operation but materially improved the chances of recovery if pulmonary and cardiac complications followed it.

Dr. F. W. COLLINGWOOD related a case of death occurring immediately after discontinuing the anæsthetic.

Patient was a woman about 45 years of age, operated upon for fibroid of uterus. After removal of the fibroid she was seen to have difficult respiration and appeared slightly cyanosed. Pulse small, 90 per minute. He (Dr. Collingwood) was called to see the case as a junior house-surgeon was administering the anæsthetic, and the surgeon thought that an overdose of anæsthetic might have been given. This, in his (Dr. Collingwood's) opinion, was not the case, as there was slight lid reflex and the pupils were only moderately dilated. The operation was rapidly concluded, but the patient expired about fifteen minutes after return to the ward. A post-mortem examination by an independent pathologist showed that the left iliac vein had been pressed upon by the fibroid removed, and still contained a piece of ante-mortem clot; the rest of the clot formed in the iliac vein had apparently become detached on removal of the fibroid at the operation, and had passed up to the right side of the heart, broken up in its passage, and several pieces of clot were found lodged in the branches of the pulmonary artery in both lungs.

## Section of Anæsthetics.

President — Dr. A. L. FLEMMING.

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### DISCUSSION ON CORONERS' INQUESTS—THE CLASSIFICATION OF DEATHS UNDER ANÆS- THETICS AS VIOLENT OR UNNATURAL.

Dr. J. H. CHALDECOTT.

THE invitation to open this discussion was prompted by a letter from me on the subject which was published in the *Lancet* a few weeks ago; for the title of my paper I am indebted to a critic in the lay press, who remarked that I was evidently unacquainted with the section of the Coroners' Act which says:—

“On report of death the coroner, if he has reason to suspect the death to be a violent or unnatural one, shall hold a public inquest.”

And the writer went on to say: “And death from chloroform poisoning, whether immediate or delayed, is obviously unnatural.”

I shall endeavour to show, first, that in view of the attitude of coroners towards other surgical deaths it is *not* obvious that all anæsthetic deaths are unnatural, and, secondly, that it would be to the public advantage if inquests were *not* held on all anæsthetic deaths as a matter of routine.

Whatever points of difference may arise in this discussion there are two upon which I am sure we shall all agree—that there is nothing in our work which we have any wish to conceal, and that this discussion is not prompted in the slightest degree by any antagonism or resentment towards coroners, who invariably treat members of the medical profession with great consideration. Its object is to invite expressions of opinion as to whether the present interpretation of the Coroners' Act in reference to anæsthetic deaths is the best possible for the community at large; and, if not, how it can be improved.

Laws are made and administered for the welfare of the public, and if it can be shown that the holding of these inquests as a matter of routine is of any material advantage to, or adds to the safety of, the community at large, it would clearly be the duty of the medical profession to endure with resignation any inconvenience and strain which falls upon them in consequence, but if it can be shown that it is a danger rather than a safeguard to the public, there can be no reason why we should hesitate to complain of the unnecessary burden which it places upon us.

What do you suppose influences coroners in regarding deaths from, say, surgical shock as natural, and in not holding inquests upon them? It seems to me that their line of thought must be that if a surgeon operates and the patient dies, the death has been caused, or at any rate accelerated by an

[April 20, 1923.]

act which was intended for the patient's good, was undertaken with the patient's consent, and formed a part of the ordinary and natural duty of a qualified and registered medical practitioner. Taking that view the coroner does not suspect the death to be violent or unnatural, and does not hold an inquest. Whatever the explanation is, the fact remains that deaths which occur from purely surgical complications, even after operations of expediency, are not made the subject of coroners' inquests, which is perfectly right and proper; but why should coroners not exercise the same discretion in anæsthetic deaths, when the anæsthetic has been given by a qualified medical man for a legitimate and perfectly natural purpose, and with the consent of the patient? The fact that it is not the practice of coroners to regard surgical fatalities as violent or unnatural, proves at any rate that they have the power, if they think proper, to treat anæsthetic deaths in the same way, so that disposes of the contention that they have no option in the matter.

We may now justly inquire, what *advantages* does the present system confer upon the public? Possibly some other speakers this evening can throw more light upon that side of the question than I can. With every wish to put the matter fairly, I have been much puzzled, and have sought information from all kinds of people, but the only advantage that occurs to me, or that has been suggested to me by others, is that when the relatives or friends are dissatisfied a public inquiry clears things up. With this I cordially agree, and it should be applied not only to anæsthetic deaths, but to all deaths occurring under medical treatment in connexion with which the relatives of the deceased think they have cause for complaint, or when information has been given which leads the coroner to suspect that there has been negligence, carelessness or irregularity of any kind. I may be wrong, but my impression is that these conditions occur very rarely, that the relatives are almost invariably satisfied that everything possible has been done, and that a public and formal inquest is usually as distasteful to them as it is to us.

Of the *disadvantages*, the most obvious to anyone who is in daily touch with surgical work is the terror of anæsthetics which the newspaper reports of these inquests create in the public mind. They are read by thousands of people who do not appreciate the fact that an enormous number of administrations take place daily, and even hourly, and who consequently fail to recognize that the percentage of fatalities is infinitesimal. This nervous apprehension is detrimental in two ways: it greatly increases the patients' nervous and mental strain immediately before operation, and lowers their resistance to shock; and, what is even more important, it frequently causes them to postpone having necessary operations done until their prospects of cure have been seriously impaired by delay. I can say with truth that I have seen many such cases where fear of the anæsthetic has prevented patients from availing themselves of surgical aid until too late.

There is another ill-effect which reacts upon the patient, and that is the additional strain which these inquests impose upon the nerve of the medical men themselves. I doubt if this is entirely negligible even in the case of senior men, but in the case of men who are on the threshold of their career the effect is much more keenly felt, and in that connexion I should like you to picture to yourselves for a moment a scene which is fairly familiar to those of us who work in general hospitals. A patient is admitted in the night, desperately ill with advanced intestinal obstruction, and the anæsthetist on emergency duty (probably a comparatively young man) is called upon to administer an anæsthetic for an operation which is at the best a forlorn hope.

It is quite possible that he may have several such cases within a few days. Suppose the first one died on the table; is it likely to steady his nerves when a crisis arises in his next serious case, if it flashes across his mind, as it inevitably must, that for the second time in a week he may have to attend before the coroner to justify his administration? Is it—can it be—to the patient's advantage that the anæsthetist should have this additional and unnecessary strain at a moment when a life depends upon his coolness and resource? There can be only one answer to that question. In a case of this kind the glaring anomaly is that if the surgeon and anæsthetist had shirked their duty and had allowed the patient to die without any attempt being made to save him by operation, the death would have been regarded as natural, but that because they strained every nerve to save him and in spite of their efforts he died on the table, his death would be suspected as being violent and unnatural, and a public inquest held.

I once saw a pulmonary embolism occur in the middle of an operation; the operation was nevertheless completed and the patient put back in bed, where she recovered consciousness, and died three hours later. The cause of death was certified as strangulated umbilical hernia three days; (operation)—pulmonary embolism, three hours. The certificate was accepted as one of natural death, and no inquest was held. Had the patient not recovered consciousness, death would in all probability have been attributed to the anæsthetic. The operation killed the patient, so death was natural; had the anæsthetic—which was an essential part of the operation—killed the patient, her death would have been unnatural, and a public inquiry would have been held. As Shakespeare says: "Is this law? Ay, marry is it! Crowner's quest law."

It may be argued that these cases I have just described are extreme instances, and that my remarks would not apply to operations of expediency when a fatality had been absolutely unexpected, and it may be asked: should not that kind of death be suspected as being violent or unnatural? I cannot help thinking that unless there is *prima facie* evidence of negligence or carelessness there is no reason to suspect it of being either violent or unnatural. The whole proceeding is a legitimate attempt to benefit the patient; it is undertaken by a qualified medical man, licensed by the State to deal with conditions of life and death, and it is difficult to understand why such death should be regarded as more unnatural than any kind of sudden death which occurs while the patient is under medical or surgical treatment. In any other such case the medical man is trusted to give a certificate of the cause of death, and it is accepted. Why not in the class of case under discussion?

If one branch only of medicine and surgery is singled out in this way, and any death occurring under it is regarded with suspicion, it is not surprising that the administration of anæsthetics is the least popular of a doctor's duties and that many medical men have the greatest possible dread of being called upon to undertake an administration, not from any timidity or diffidence as to their capability, but because they are unwilling to risk the publicity and consequent damage to their reputation should they be unfortunate enough to have a fatal case. In hospitals it certainly has the effect of making house officers very shy of this work, and discourages them from availing themselves of all the opportunities for anæsthetic experience which their office affords them. This is, of course, an additional disadvantage to the public.

How inquests can be harmful in some cases, and useful in others, is well illustrated by two anæsthetic deaths which have occurred quite recently. The first was that of an aged man who died during an operation after he had been

under the anæsthetic for some time. The autopsy disclosed well-marked fatty degeneration of the heart which made it obvious that he might have died suddenly at any time; in this class of case a post mortem without a public inquest would have been quite sufficient, and would have done no harm, whereas the result of the inquest was a sensational report in the evening press, with the alarmist heading of "Operation Risk: Heart Disease that escapes Diagnosis." It would be difficult to imagine anything more mischievous and misleading. The impression which this would convey to many people is that a hitherto unrecognized danger had been discovered, which was an addition to the already well-known risks attendant upon operations, whereas the truth is that it is an exceedingly common condition which is frequently the cause of sudden death, but in the possession of which many patients are anesthetized without giving the administrator the slightest anxiety.

The second case was one in which the patient lost her life from the afferent and efferent tubes of a Junker's chloroform apparatus having been transposed. This was a typical example of an accidental death, and one in which an inquest was clearly called for, in order to ascertain whether or not the mistake was due to any negligence or carelessness. In a case of this kind the coroner could not do otherwise than suspect that the death was violent or unnatural.

The objections which I have enumerated apply with even greater force to the holding of public inquests upon deaths which occur several days after operation, such death being attributed to the indirect or delayed effect of the anæsthetic. It is in holding inquests upon this class of case that the greatest inconsistency is shown. When one bears in mind the large number of fatal complications which may follow operations, one cannot refrain from asking, what good purpose is served by an inquest on a case of delayed chloroform poisoning, which would not be equally well served by one on a death from shock, from secondary hæmorrhage, from pulmonary embolism, or for the matter of that, from ether bronchitis? In all these cases death has occurred from complications which must occasionally crop up in surgical work—complications, which taken together, make up the sum total of operation fatalities and represent an all-round percentage of mortality which is exceedingly low. Why, therefore, pick out one complication, and suspect it to be violent or unnatural? I am not going to trouble you with statistics, but it is common knowledge that the mortality of surgical operations becomes less and less. If any operation or particular method shows a high mortality that operation or method is quickly altered or abandoned, and for years surgical reports have shown steady and continuous progress. Would that have been so if every surgical death had been made the subject of public inquiry, and the surgeon had been called upon to explain and to justify his technique to a lay tribunal? Surely the effect would have been most disastrous, and would have tended to cramp surgical progress at every step. Suppose that the comparative safety of two methods of a particular operation instead of being settled as they are now by those responsible for them, by a comparison of each other's results, were threshed out in a coroner's court. Would such a farce be in the public interest? Now for a moment let us apply that illustration to anæsthetics. Take the question of the selection of the anæsthetic in any particular case. Supposing I were to submit to you to-night a certain case and I described to you, in detail, the type of patient, his physical signs, and the nature of the operation, and were to ask for an expression of opinion from you as to what anæsthetic you would give, and how to give it. My

inquiry would produce various answers, all of which would be good, and each of which would at any rate represent the studied judgment of the man who gave it, and yet, at a coroner's inquest a complicated matter of that sort has to be settled as right or wrong by a single question and answer!

For someone who has never seen the patient during life to suggest to an anæsthetist in public, after a fatality, that he gave the wrong drug, seems to me not only futile, but unjust.

The impressions which are conveyed to the lay mind by public discussions on the relative safety of chloroform and ether are almost invariably misleading, and I suggest that such points are outside the functions of a coroner's court. These discussions can do no good and from a medico-legal point of view—the only point of view which is relevant at an inquest—they can have no bearing upon the verdict.

The growing popularity of ether in this country is in the opinion of most of us much to be commended, but anything resembling an official lay condemnation of chloroform and its consequent rejection in suitable cases, would be nothing short of a calamity. This critical attitude towards chloroform on the part of some coroners is already beginning to cramp the teaching of chloroform administration in the hospitals, and is, I am convinced, destined to do much more harm than good. The old motto that "One man's meat is another man's poison" was never more aptly applied than in the case of anæsthetics. An unqualified expression of opinion in court that ether is safe and chloroform dangerous is quite inaccurate. We know that in some cases, with some patients, and in the hands of some administrators, the position may be exactly the opposite. The outstanding fact is that the best judge of the safest method on any particular occasion is the man who has to take the responsibility of the administration, and surely it ought to be assumed without criticism or inquiry that the administrator, whatever his choice may have been, acted according to his best judgment and in the best interests of his patient.

There is an old ballad, one couplet of which runs:—

"A little bird whispers, perchance you may swing,  
For a crowner's quest is a mighty queer thing."

This is, perhaps, an extreme view! But there is no disguising the fact that in every one of these inquests the anæsthetist appears to be on his trial, and is at the great disadvantage of sometimes being called upon in cross-examination to explain complicated technical points to a tribunal whose knowledge of the subject is negligible. This is a most unnecessary and unfair position in which to place him, and the only gratifying feature of it is that so far, at any rate, as I can remember, no case has been heard which has disclosed any negligence on the part of the anæsthetist.

There can be only two reasons for holding these inquiries: First, to find out if anyone is to be blamed for the death; and secondly, to elicit some clinical fact which may be of assistance in diminishing anæsthetic mortality in the future. The first is really the prime function of a coroner's court, but in these cases it is unnecessary to exercise it unless there is ground for suspicion. For the second, a coroner's court is the worst possible form of tribunal, and a substitute should be found. The medical profession, in this, as in every other branch of their work, are anxious to learn from their failures, and they would eagerly welcome any form of inquiry which is likely to be



of practical use. Such inquiry should be official, but private; it should be carried out by a committee, some members of which should be experts, who know enough of the subject to view the administration of anaesthetics from the point of view of the requirements of modern surgery. Such committee should be appointed by the Ministry of Health, and should have the power to call for all records of the fatal cases—clinical, operative, and post-mortem. In this way every point which might be useful in the future could be elicited and recorded without the danger of producing false alarms through indiscreet newspaper reports; and at the end of each year the committee would issue a report to the General Medical Council, which could be published in the medical press, containing any suggestions which they thought might prove valuable.

If this were done it would relieve coroners of holding inquests upon any of these fatalities, except those which present the special features to which I have alluded, and the interests of the public would be better safeguarded, not only by the fact of the inquiries being held by a tribunal competent to deal with the technical side of the question, but also by the absence of publicity. Now that we have a Ministry of Health it ought not to be a difficult matter for this to be arranged, and I venture to suggest to the Council of this Section that they might consider the advisability of seeking an interview on the subject with the President of that Department.

I do not agree with the prophecy of my above-mentioned critic that an agitation to alter the system will meet with little sympathy either in Parliament or from the public. I feel confident that if it is clearly put before them, the public would be quick to grasp the fact that their interests in this matter are inextricably interwoven with ours; that anything which tends to cramp teaching, and to foster in the minds of young practitioners a fear of acquiring anaesthetic experience, must be a public danger rather than a safeguard, and that they would gladly support an effort to reform a system of inquiry which however well adapted for murders, suicides, and ordinary accidents, is quite unsuitable for the majority of deaths in connexion with operations.

#### MR. I. HAMILTON BEATTIE

said that he fully agreed with the opener of the discussion on the subject of chloroform. In order to illustrate the unsatisfactory nature of the present procedure for investigating deaths at operations, he would give the notes of two cases occurring in his own practice during the last year, both in the same coroner's area.

*Case I.*—A ruptured ectopic was admitted to hospital desperately ill. After administration of subcutaneous saline it appeared possible to operate. Gas and oxygen (without ether) was given by Boyle's apparatus. On opening the peritoneum circulation and respiration suddenly ceased. The surgeon massaged the heart through the diaphragm and reported this manoeuvre ineffectual. Meanwhile saline and pituitrin had been given without result. Adrenalin was now injected direct into the heart, which immediately started beating again. Colour returned and pulse became strong, though artificial respiration was required for a further ten minutes. The operation was rapidly performed and the patient put to bed, where she developed Jacksonian fits which occurred continuously until her death six hours later. She did not become conscious, but no inquiry was held; though it would certainly be in the public interest to discover why the patient was not brought to hospital earlier.

*Case II.*—Diathermy for inoperable malignant disease of symphysis menti. Colonic oil-ether was given at surgeon's request, as the mouth was extensively involved in



the disease. All went well, pulse and respiration remaining perfectly steady throughout the operation, which took about forty-five minutes. Three hours later the patient died, and an inquest was held because he had not recovered consciousness.

A particularly unfair point was that at an inquest the anæsthetist was always asked how many previous deaths had occurred in his practice, while no such question was put to the surgeon.

#### Dr. MENNELL

said that everyone must admit Dr. Chaldecott had put his case very fairly and in most moderate language, but he did not think he had brought out clearly enough the fact that deaths under anæsthetics could be divided into two classes :—

(1) Deaths due directly to the anæsthetic.

(2) Deaths due to the disease for which the operation was being performed.

Why should the anæsthetist be called to the coroner's court for such cases as rupture of thoracic aneurysm or damage to the floor of the fourth ventricle causing death while the patient was under an anæsthetic? Such deaths had no relation to the administration of the anæsthetic. Again he was interested to hear Dr. Chaldecott's views about a committee, as at St. Thomas's such a committee had been in existence for the last three years. It was a sub-committee of the general committee and consisted of a surgeon, pathologist and anæsthetist appointed to inquire into every death during the course of anæsthesia, and to report when necessary to the main committee. Every anæsthetist concerned in the death had to fill up three forms which were sent at once to each member of the committee, who met as soon as possible and if they thought fit questioned various people concerned. In this way the information gained was very striking, but as the question of the relative advantages of different anæsthetics was not under consideration details were not given. Scientific research was hampered by the fact that the body was removed to the mortuary, but on more than one occasion, owing to the kindness of the pathologist acting for the coroner, specimens had been taken; the other great difficulty to be contended with was the nervousness of the house officers, and care had to be taken not to intensify this. Such a committee worked easily and well, and gained valuable information, but of course had no official standing outside the hospital as would that committee the formation of which was proposed by Dr. Chaldecott.

#### Mr. H. R. OSWALD

(Coroner for the Western District of the County of London, and President of the Coroner's Society of England and Wales) said he hoped that anæsthetists did not think that coroners had an inordinate wish to hold inquests on the bodies of people who had passed away while under the influence of anæsthetics. In the present state of the law, however, they had no power to act otherwise, in the majority of cases reported to them. Their interpretation of the law, he submitted, was the correct one, in regarding these deaths as "unnatural, because a death due to an anæsthetic was one which arose from the administration of a poison. The contention that a fatal end to a surgical operation was also unnatural and, therefore, required an inquest, was not a perfect analogy. A surgical operation was not a poison. It was an attempt to rescue a patient from death, or to alleviate suffering, which occasionally

proved a failure owing to the difficulty of the undertaking, or the inability of the patient to endure the shock. There might be a misadventure now and then, but he believed it was a rare occurrence. Out of 925 inquests held in his district during the year 1922, there had been twenty-five on cases of anaesthesia, and in none of these had any adverse reflection been cast on the anaesthetist; nor could the speaker recall, in an experience of more than twenty years, an instance of blame being attached to the anaesthetist. On the contrary he had heard of cases where the friends of the deceased had come to the inquiry with minds very hostile to the doctor, but, after hearing the evidence, had left perfectly satisfied that he was in no way to blame. It was only after a post-mortem examination had been made that the real cause of death could be stated. From scientific considerations there was a good deal to be said for an investigation in private by an exclusively scientific tribunal, but he doubted whether it would command the same confidence among the public as an impartial inquiry in open Court, which he, were himself the anaesthetist who had acted with care and skill and to the best of his ability, would prefer to any semi-secret investigation.

#### Mr. FINUCANE

said that the real test was: (1) Was it a sudden death? (2) Was it an unnatural death? (3) Was it an accident?

Surely a death under an anaesthetic was caused by the inhalation of a poison, and was a sudden death, an unnatural death and an accident. The analogy between death from an anaesthetic and surgical shock were not in his view parallel cases, and, further, the ultimate test was, could the anaesthetist, or the operating surgeon, give a death certificate, in these cases, of natural causes? If neither of them could do this, then it followed, *ipso facto*, that an inquest must be held, quite irrespective of the legal obligations of the coroner in all such cases, under the existing law, to hold an inquest.

Inquests were also frequently demanded by public opinion and complaints of relatives, and coroner's courts had shown themselves to be not only public inquiries of a very necessary character, both in the interests of all parties concerned, but further in the highest scientific interests, both statistically and pathologically.

Were not the very differences in the profession and the professional knowledge of the respective merits of anaesthetics and dosages more or less public controversies, of which the public claimed knowledge and full investigation? and those public advantages, added to the absolute legal requirements, far outweighed any of the terrors, or strain of nerves, or alleged public alarm which might follow, and which in all cases had resulted with complete credit to the anaesthetists.

That everybody must submit to inquiries, where life was lost, as to their negligence or want of skill, seemed to him (Mr. Finucane) to follow as a natural and proper result, and certainly was a fundamental principle of the coroners' law and inquests.

#### Dr. H. P. CRAMPTON

suggested a point of view that had not yet been brought out—the importance of the skill and speed of the operating surgeon with reference to the results of the anaesthetic: for example, a severe ruptured ectopic in the hands of an expert and rapid operator might do well, but if the surgeon were not so

skilful, the patient and the anæsthetist being the same, a catastrophe might result. It was manifestly impossible for an anæsthetist to stick up for himself at an inquest to the discredit of the surgeon.

Mr. BOYLE

thought that the time had come for the Section to make another effort to get the Coroners' Act altered; he believed that the late Sir Frederic Hewitt had tried to bring this about, but so far there had been no result. As far as the coroners themselves were concerned he had the greatest admiration for them as a body of men, but he did not think that a coroner's jury was a fit and proper body to adjudicate on an anæsthetic fatality. What was wanted was a small body of say three men—a pathologist, an anæsthetist, and either a surgeon or physician—whose duty should be to hold an inquiry into all anæsthetic fatalities. They should be properly paid for their work. If this was done he felt that we would obtain a considerable amount of valuable scientific information, which, under the present conditions, was running to waste.

Mr. MORTIMER

remarked that the Report of the Departmental Committee of the Home Office, approved by the General Medical Council and various institutions in 1910, recommended *inter alia* that a *scientific investigation* should be made in any case of death under an anæsthetic in a hospital, and that a small standing scientific committee on anæsthetics should be instituted under the authority of the Home Office: but from that day to this no action had been taken.

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PROCEEDINGS  
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**VOLUME THE SIXTEENTH**

SESSION 1922-23

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SECTION OF BALNEOLOGY AND CLIMATOLOGY.



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## Section of Balneology and Climatology.

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# SECTION OF BALNEOLOGY AND CLIMATOLOGY.

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## Section of Balneology and Climatology.

President—Dr. J. CAMPBELL McCLURE.

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### DISCUSSION ON BLOOD-PRESSURE.

J. CAMPBELL McCLURE, M.D. (President).

VERY properly, alterations in the blood-pressure from the physiological normal are now regarded as symptomatic and not as constituting in themselves a disease.

This is the more certain since the routine study of the blood-pressure has shown that the physiological normal can vary within a very wide range. After exercise, short of great fatigue, the blood-pressure tends to rise—rest and exhaustion tend to lower the blood-pressure. If this rise is temporary, little heed need be paid to it. Leonard Hill has pointed out that the effect of increasing the rigidity of the arterial system is to increase the pulse effect in the more distant parts of the circulation, so that it would seem that increase in the blood-pressure is a response on the part of the arterial tree to a demand for a greater visceral pulse made, for example, by the muscles, or by the kidneys, the eliminative capacity of which has been more than usually taxed. If this eliminative strain is continued too long, degenerative changes occur in the arteries and in the kidneys themselves, and the condition, which began as an effort on the part of our intricate metabolic machine to balance itself, results in a definitely morbid state, with gross pathological changes in the tissues. We have all known cases of high blood-pressure in which experience showed that the patients had an *optimum* pressure which was greatly above the normal, and in which reduction to something approaching normal was always associated with a degree of discomfort on the part of the patient that rendered life difficult and work almost impossible. I recollect one patient who suffered from intolerable headaches in connexion with chronic renal disease and whose systolic blood-pressure was well over 200. This unfortunate man came under my care in my earlier days in medicine, when my enthusiasms were greater than my experience. I considered that the headaches might be relieved by a reduction in the pressure, and I gave the man sundry preparations of the nitrites. The object was in the main achieved; the blood-pressure had fallen nearly to normal and the medication was stopped, but the pressure continued to fall and the man died in the course of the day. I am glad that this lesson came to me early in life. I had removed a symptom without removing the cause, and the patient suffered.

It is extraordinary how capable, if not comfortable, some patients can be with a blood-pressure that seems fantastic to most people. I recollect a patient who lived for four years, and at her weekly visit to the out-patient department her systolic blood-pressure was never below 290 and frequently 300.

In a paper published last year in the *Lancet*,<sup>1</sup> Dr. Ellis and I pointed out that in certain patients who were intolerant of acids there was evidence of renal strain, as shown by rise of blood-pressure and signs of acidosis after the administration of moderate doses of dilute hydrochloric acid for therapeutic purposes, over a period of some time.

I suggest to you, therefore, as a basis for discussion that in most cases heightened blood-pressure is, primarily, one of the efforts made to obtain efficient elimination of acid waste products when their production is in excess of normal, or when there is some defect, functional or structural, in the renal mechanism.

Abnormally high blood-pressure may, of course, be associated with certain nervous states due, in all probability, to the presence in the blood of certain internal secretions in excess of normal.

The question of the association of heightened blood-pressures with cardiovascular changes I leave for others to discuss, largely because degenerative changes in the heart, aorta and other visceral arteries are compatible with a radial blood-pressure that is persistently normal or subnormal.

Low blood-pressure is quite as interesting to me as high blood-pressure, and it forms, and is found as, a part of disorders which have a very definite clinical entity. In true influenza I have noticed that a drop in the blood-pressure appears very early in the disease, and the pressure may remain low for many months after the patient has recovered from the attack. In all cases of post-influenzal asthenia which have come under my notice, low blood-pressure has been present, persisting until the asthenia has passed away. Again, in neurasthenia associated with gastro-intestinal atony the blood-pressure is low in the great majority of cases. In this connexion, it is interesting to record the fact that among the cases of gas poisoning which occurred in the later stages of the war patients in whom tachycardia and gastric atony persisted after the acute toxæmia had passed off, all those I have had the opportunity of examining, with very few exceptions, gave a blood-pressure reading much below the normal. Experiments conducted with the German gas used at that period were interesting. Professor Joseph Barcroft, of Cambridge, told me that in animals poisoned with this gas it was found impossible to stimulate the stomach through the vagus.

In active pulmonary tuberculosis a low blood-pressure is the rule.

During the past three years I have examined a large number of asthenic boys and young adults who presented no sign of any definite disease; young people who were easily tired, physically and mentally; "faddy" over their food; prone to gastro-intestinal upsets with distension of the stomach and colon; pallid, with atonic muscles; subject to unusual tachycardia on exertion and emotion; presenting, in other words, the picture of "*Die asthenische Krankheit*" of certain German observers. All of these had a blood-pressure that was definitely subnormal.

I am beginning to believe that low blood-pressure is an indication of a constitutional state, either congenital, or acquired as the result of certain toxæmias, which is evidenced by slow development except along emotional or artistic lines, the patient showing a distinct tendency to easy nerve exhaustion, and being very prone to fall a victim to infections such as tuberculosis.

By inference, it is not, perhaps, going too far to attribute such signs as gastro-intestinal atony and tachycardia to a lack of balance between the

<sup>1</sup> *Lancet*, 1921, ii, p. 271.

influences exercised by the vagus and sympathetic systems; to presume, in certain cases, a lack of vagus control, in others an excessive sympathetic influence.

Many of the adult patients in the groups which I have described above have shown, from time to time, a tendency to feel unusually tired after periods of strain, and when they were examined during this time of exhaustion the blood-pressure was again found to be low. I have noticed that a pint of stout or a glass of port is the best restorative one can give, and raises the blood-pressure to the average normal level of these patients.

A blood-pressure which is too low seems to me to depend on a nervous condition due to toxæmia, which may be primary or secondary to an endocrine deficiency and is often associated with a difficulty in calcium metabolism. In the neurasthenic cases associated with gastro-intestinal atony, clinical experience suggests that the condition may be produced by a primary toxæmia of intestinal origin, or a direct exhaustion of the vagus centres in a patient whose endocrine balance is defective, either congenitally, or as the result of some acute infectious disease, such exhaustion resulting in gastro-intestinal atony and subsequent toxæmia of intestinal origin.

I will close with a word of caution regarding the low blood-pressure so often found in active pulmonary tuberculosis. While a systolic reading in an adult of 110 or lower should always make one suspect tuberculosis in the absence of other definite signs, active pulmonary tuberculosis is sometimes found in association with a systolic reading of 140, 150 and even up to 170. Such higher readings tend to make me give a favourable prognosis. We must not fall into the error of an enthusiastic practitioner in London who having recently purchased a sphygmomanometer refuses to diagnose pulmonary tuberculosis in any case when blood-pressure is normal or higher than normal.

#### Dr. HALLS DALLY.

Until recent times, persistent rise in the arterial blood-pressure has for the most part been attributed to such vascular, cardiac or renal changes as could be expressed in such definite pathological terms as thickening of arterial walls, hypertrophy of heart and impermeability of kidneys. Our range of vision is now wider, and we are thus enabled to draw a broad line of distinction between two main types, the functional and organic, a division which will be found of considerable service in arriving at more exact views as to management of such cases, and which differs materially according to the type involved. Functional hypertension connotes greater or less temporary physiological disturbances arising from conditions which unduly stimulate the "pressor mechanism," whilst in the organic class there are present mechanical and pathological states which lead to persistent rise of blood-pressure.

It is, nevertheless, probable that organic arteriosclerosis not infrequently begins as functional hypertension, but time does not permit me to enter into discussion of the interplay of sympathetic activity and endocrine imbalance with the influence of the psychic factor on both. It is enough to say that, in a case of hypertension where no cardiovascular or renal factors are apparent, we must attempt to discover which glands, if any, are at fault, and of these the chief are the thyroid, gonads and pituitary, tending by abnormality in function to irritation or over-stimulation of the adrenals, between which and the above there exists an intimate correlation.

Some forms of high blood-pressure may be of the nature of an anaphylactic

phenomenon and indicate reaction of the organism against certain protein toxins. Here it becomes necessary to search for and remove the cause of the particular sensitization.

A large number of cases of hypertension are essentially toxic in origin, and of vasoconstrictor effect, and the opposite condition of hypertension may also be brought about by the absorption of vasodilator toxins. Oral sepsis and gastro-intestinal stasis should, therefore, always be looked for and appropriately dealt with. The low pressure cases, especially when associated with notable arteriosclerosis, are of much interest, and further work is required to elucidate the aetiology of these.

A point to which I specially desire to draw attention is the importance of estimating in all cases the diastolic pressure as well as the systolic, the reason being that the diastolic pressure is a charge which the arteries must constantly bear, and from which they cannot escape. The diastolic pressure, therefore, is a constant charge, whilst the systolic pressure represents only an intermittent supercharge.

Supposing the interval between the systolic crest of the pressure wave and the bottom of the diastolic notch were always the same, it would not matter which reading we took, and we might then safely rely upon systolic pressures alone. We all know, however, that the amplitude of the pulse wave varies enormously in different individuals and indeed in the same individual at different times. In other words, the relation between systolic maximum and diastolic minimum is not identical. Suppose, for example, in one man the systolic pressure in the brachial artery to be 140 mm. Hg and the diastolic pressure to be 80 mm. Hg; in another the systolic to be 120 and the diastolic 100. Which of these two has the higher blood-pressure, and in which is the artery undergoing the greater strain? If we are content to record systolic pressure alone, we should say that the man with a systolic pressure of 140 ran the graver risk. If we regard the diastolic alone, we should say that the artery of the second man was exposed to the greater strain, but if we take the mean pressure, we see that the strain is equal in the two cases.

Let me give you as a further illustration two actual cases: A patient of mine suffering from aortic regurgitation yielded a systolic pressure of 210 mm. Hg. Another, a case of granular kidney, gave a systolic pressure of 180 mm. Hg. Had I regarded the systolic pressures alone, I should have said that the aortic case had the higher blood-pressure and that his arteries were undergoing greater strain than those of the patient with granular kidney. Records of the diastolic pressures, however, put an entirely different construction on the matter. The diastolic pressure of the aortic case was 70 mm. Hg, whilst that of the renal case was 140 mm. Hg, i.e., the patient in the renal case had his arteries kept constantly on the stretch by a minimal pressure of 140 mm. Hg, which during systole rose to 180, whilst in the aortic case the diastolic pressure was only half this amount, and it was solely during the brief interval marked by the upper part of the sharp systolic peak that the pressure reached a notable elevation, whilst during diastole the arteries were far less stretched than normally. Hence, from the practical point of view, I would strongly urge a consideration in all cases of the three factors of systolic, diastolic and pulse pressures. The pulse pressure is of great importance in assessing the reserve power of the heart and in estimating its efficiency, and should not be overlooked.

There are still many gaps in our knowledge. What most general practitioners want to know is under what conditions and within what limits it is

justifiable to reduce a high or to increase a low pressure, and how this is best brought about. Here it is very difficult to lay down hard and fast lines, and each case has to be dealt with on its merits. We have always to be content with guiding rather than driving, and if one errs, to err on the side of doing too little rather than doing too much.

Just one more point as to prognosis. Dr. Graham-Stewart, of Margate, in the *Practitioner* for September, 1921, stated that it is almost impossible in cases of fully developed chronic nephritis to lower the systolic blood-pressure except in a very temporary way. Indeed so much so is this the case that he has been led to believe this failure to reduce abnormally high pressures as being almost pathognomonic of the presence of renal disease. In differentiating hyperpiesis from early interstitial nephritis he regards the same point as useful in diagnosis as well as prognosis, although the one condition may merge into the other. Frequent observations have led me to support these views and I mention them in order to stimulate others to work on these lines.

#### Dr. STRICKLAND-GOODALL.

Discussions on important subjects such as blood-pressure are interesting, not only on account of the facts elicited in connexion with them, but also on account of the very different standpoints from which different clinicians approach the subject.

As the President has clearly pointed out, blood-pressure is simply an expression of some general underlying condition; its treatment therefore should essentially depend upon the recognition and appreciation of this underlying condition. Systematic attempts to *lower raised blood-pressure* or to *raise lowered blood-pressure*, without due appreciation of the cause, are not merely wrong but frequently dangerous. What, for example, could be more pernicious than an attempt to raise the lowered blood-pressure which occurs in influenza and diphtheria, and which is due essentially to a weakened and poisoned myocardium, or to lower the raised blood-pressure of aortic regurgitation, where the raised blood-pressure is such an important asset in the maintenance of an efficient circulation?

Many classifications for high or low blood-pressure cases have been suggested, but if one bears in mind that arterial blood-pressure depends on three prime factors, namely: (1) the force and frequency of the heart (this involves the myocardium and its nervous mechanism); (2) the peripheral resistance (which consists essentially of the vasomotor mechanism and the arterioles); (3) the output, i.e., the quantity of blood ejected at each contraction—it would seem logical to divide the blood-pressure cases into three main groups, namely, those in which the condition depends primarily on the heart, those in which it depends primarily on the peripheral resistance, and those in which the output is chiefly involved. In most blood-pressure cases there are probably several factors; in aortic regurgitation, for example, one has increase in the force of the heart, increased output tending to *raise* the blood-pressure, and relaxed peripheral resistance tending to *lower* it. In Graves' disease the blood-pressure is triphasic, there being first an initial rise due to vasoconstriction, followed by a prolonged fall due to peripheral relaxation, followed in turn by a tertiary rise usually associated with a recovery in peripheral tone.

In mitral stenosis the blood-pressure is usually low, owing chiefly to the diminished output and poor ventricular contraction.

Of the different groups of cases that which I have termed the myocardial

## 6 Strickland-Goodall: *Discussion on Blood-pressure*

group is the one in which thoughtless treatment is liable to do the greatest harm. Although, generally speaking, it is the underlying condition which must be taken as the index for treatment, and not the actual blood-pressure itself, one must bear in mind that there are certain definite dangers associated with at any rate an abnormally high pressure. One has for example to face the fact that a persistent pressure round about 300 is very liable to result in either (1) hæmorrhage, especially either into the eye or brain, or (2) cardiac dilatation with failure.

It is essential in such cases, *irrespective of cause*, to lower the pressure temporarily by such means as bleeding, baths, &c., with a view temporarily to resting the myocardium and reducing the arterial strain. It is highly probable that the treatment accorded to blood-pressure cases at such resorts as Nauheim, Bath, Harrogate, &c., are efficient, popular and useful for these reasons.



## Section of Balneology and Climatology.

President—Dr. J. CAMPBELL McCLURE.

### DISCUSSION ON THE VAGUS AND SYMPATHETIC NERVES AND THEIR RELATION TO CLIMATE AND HYDROLOGY.

Dr. C. F. SONNTAG (London).

A KNOWLEDGE of the vagus and sympathetic nerves in health and disease is of great importance to the spa physician, and his interest in them is two-fold. In the first place many of the patients who come under his care are suffering from a vago-sympathetic disturbance in one or more organs; in the second place the hydrotherapeutic measures, and the climate of the spa in which he resides, exert a beneficial or injurious effect through these nerves. Unfortunately the text-books do not deal with the subject in the manner most suitable to the spa physician, and he has neither the time nor the opportunity to keep in touch with the growing literature. The aim of the present discussion is to bring together the views of those who have devoted special attention to different aspects of the subject, thus providing the practitioner with a useful guide to treatment. I have been requested to open the discussion, and my remarks will deal mainly with the foundation subjects, comparative anatomy and comparative physiology. They are based on observations made in the Anatomy Department of the Zoological Society of London during the past four years. The detailed accounts of the nerves in various mammals have been published in the *Proceedings of the Zoological Society* for the years 1921-23.

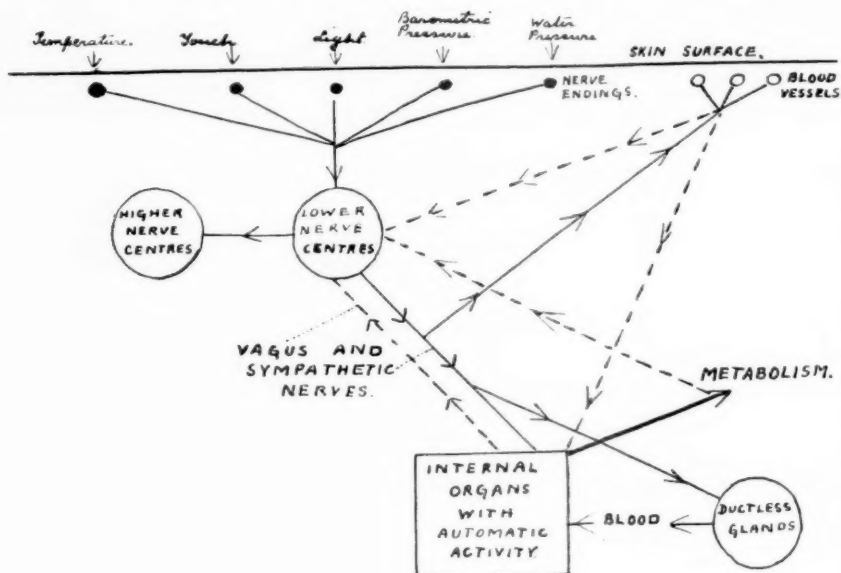
Many of the effects of climate, which has greatly modified the character and distribution of animal life in the past, are due to the action of stimuli of temperature, light and variations in barometric and hydrostatic pressure on the cutaneous nerve-endings. And the effects of hydrotherapeutic measures are due to physical agents acting on the same structures. Impulses are set up which travel by way of the afferent nerves to the lower nerve centres. Some reach the higher centres, and Professor Elliot Smith believes that the large size of the human brain is partly due to the existence of innumerable nerve-endings in the skin. Fortescue Fox states that these impressions influence mental processes to a considerable extent. Fresh stimuli pass out from the nerve centres in different directions:—

- (1) By way of the vagus and sympathetic nerves to the organs.
- (2) Through the sympathetic nerves to the cutaneous blood-vessels and

endocrine organs. The changes in the calibre of the cutaneous arterioles produce secondary changes of an opposite nature in the viscera.

Afferent impulses also pass from the various organs to the nerve centres through the vagus and sympathetic nerves. The result of all these actions is a change in metabolism, which varies in character in accordance with the nature of the climatic and hydrotherapeutic stimuli. The various routes for the impulses are shown (*see figure*).

It is common knowledge that many of the internal organs have an automatic activity which can be altered in many ways by extrinsic stimuli acting through the vagus and sympathetic nerves; and the effects of climate and baths are partly or entirely due to such impressions. If an organ is deranged these impressions will keep up the disordered action, and they are



ably assisted in this harmful work by stimuli from other organs. During the war we treated cases of nervous and cardiac irritability resulting from shell-shock and other causes by immersing the patients in a sedative pool bath, the temperature of which was that of the nerve-endings in the skin. Sooner or later the symptoms disappeared, and it seemed as if vagus action were favoured. Of course other conditions assisted in promoting recovery, but I believe that the water cut off the varying impressions which play continuously on the cutaneous nerve-endings.

When the vagus and sympathetic nerves in man are compared with those in lower animals it is seen that:—

- (1) Man has the most complex sympathetic system.
- (2) The abdominal parts of the vagus nerves are more complex in some mammals, for their digestive organs are highly specialized.

In all mammals the vagus and sympathetic communicate in the neck, and branches which usually come from the vagus may be given off by the sympathetic (e.g., *Dendrolagus ursinus*). Of course the vagus filaments have passed through the communications. The reverse is the case in some carnivora, in which the entire sympathetic supply to the heart is contained in branches of the vagi.

Before considering the nerves in detail it is necessary to emphasize three points in regard to the actions of hydrotherapeutic measures. In the first place there are very few local effects, most of the induced changes being reflex in character. In the second place all applications, no matter how weak or transient they are, produce a change in the rate and force of the heart. In a former paper read before this Section I showed the charts recording the actions of different kinds of baths on the pulse. In the third place localized applications induce changes in the internal organs reflexly connected to the area of skin treated.

The effects of full baths are so well known that no description is needed here.

The autonomic nervous system is divided into three parts, each of which is called into play by certain hydrotherapeutic measures. The bulbar autonomic is influenced by bathing the face and, possibly, by throat compresses. The thoracic outflow, or sympathetic proper, is aroused by the majority of general and local treatments; and the sacral part is influenced by hip baths, douches and irrigations.

The application of cold water to the face, particularly when it is applied as a spray, produces a gasp followed by a pause in respiration. The terminals of the trigeminal nerve are excited and a reflex is induced by way of the bulbar autonomic apparatus; the impressions are carried to the lungs by filaments in the vagus nerves, and vaso-motor changes are also induced in the mucous membranes of the head. Fredericq observed how respiration is arrested in diving birds when water is injected into the nostrils; and it is possible that the admission of water into the blow-holes of whales may stop the respiratory movements when the animals are submerged. In *Ornithorhynchus* the skin of the bill, which is supplied by the enormously-developed trigeminal nerves has peculiar tactile organs which, it is believed, enable the animal to know the depth of the water in which it is immersed.

The cervical parts of the vagus and sympathetic nerves are of interest to the hydrologist in connexion with the application of throat compresses, which are so beneficial in pharyngitis and laryngitis. The skin of the neck is supplied by C2, C3, and C4. These nerves communicate with the ganglia of the vagus and sympathetic, but the stimuli do not pass through the latter, which are grey rami communicantes. The detection of the path I must leave to the physiologists present. If, however, the application touches the face it excites the mandibular division of the fifth nerve and acts through the bulbar autonomic system.

The vagus, glossopharyngeal and sympathetic nerves form the pharyngeal plexus, which is closely related to the tonsils and vessels which form Waldeyer's lymphatic ring. And the reflex phenomena which are present in adenoids are probably due to irritation of the nerves forming the plexus. Moreover it has recently been shown that infection from the appendix produces lymphangitis in the plexus of lymph-vessels surrounding the solar nerve plexus, and induces various reflex phenomena.

The inferior cervical ganglion of the sympathetic sends a thick nerve or a

bundle of filaments along the vertebral arteries; and it sends vaso-motor nerves to the arms which are called into play by all arm baths. Branches also run to the cardiac plexuses, but in some animals these nerves lose themselves in the rich plexuses which accompany the branches of the aortic arch. In a chimpanzee I observed it sending a well-marked plexus of nerves to the thyroid gland along the thyroidea ima artery. Some of the vasomotor nerves to the arms arise from the fused inferior cervical and first thoracic ganglia in many animals. In *Canis thous* a large swelling on the vagus gives off the thoracic gangliated cord and sympathetic nerves to the fore-limb.

The sympathetic innervation of the heart varies considerably. In man branches come from each of the cervical ganglia, but I did not observe a cardiac branch arising from the superior ganglion in any other mammal; a cardiac branch arose from the middle ganglion in each of the few mammals in which that body occurred; and the inferior ganglion always gives off cardiac branches. The filaments reach the heart in one of three ways; they proceed directly to the cardiac plexuses; they are contained entirely within the vagus; or they get lost in the rich plexuses of the heart, and of the aorta and main vessels arising from it.

When the gangliated cords of the sympathetic in man are compared with those in other mammals several interesting differences are observed. Man has a ganglion corresponding to each rib, and filaments run from most of the ganglia to form the splanchnic nerves. In many mammals the ganglia are not so numerous, and in several species the cords become the splanchnic nerves and end in the solar plexus; in these animals the abdominal cords spring from the solar plexus. In the cetacea the ganglia, which are very large, have the usual rami communicantes, but nerves are also given off to the rich rete mirabile within the thorax. All these elements form a complex neuro-vascular mechanism, which is called into play when the animals dive or rise to the surface of the ocean. When they sink the pressure on the skin is enormously increased, and when they ascend again the pressure falls. This greatly subdivided arterial network receives blood from the skin when the animals dive, and the blood-pressure will not rise greatly. When a man dives into cold water the cutaneous arterioles are constricted, and the arterial tension is so increased that a hæmorrhage may occur if the vessels are brittle. If his arterial system were as divided as that of the whale the tension would not be great.

The cardiac branches of the vagi differ in man from those in lower mammals, for upper cervical cardiac branches are uncommon in the latter. In all mammals the thoracic parts give off cardiac nerves, and the left recurrent laryngeal nerve supplies twigs to the heart. It is, however, uncommon to find cardiac branches arising from the right recurrent nerve.

The anterior pulmonary branches of the vagi arise separately or as bifurcations from cardio-pulmonary nerves. The posterior pulmonary plexuses are formed by a breaking up of the vagus trunks, or, as is most frequently the case, by separate branches from the vagi. After the pulmonary branches are given off the vagi contain very few medullated fibres.

In the posterior part of the thorax the vagus nerves form a more or less complex œsophageal plexus. In the simplest form the two nerves send twigs to the œsophagus; but there is a more or less intricate fusion of the nerves themselves or of branches over the front of the œsophagus.

The vagus nerves end in several ways in the abdomen, but one of them

always runs along the lesser curvature of the stomach. The other terminates in one of the following ways:—

(1) It ends mainly in the stomach, and it sends a communication to the solar plexus.

(2) It ends in one of the cœliac ganglia after giving off gastric branches.

(3) It communicates with the cœliac ganglia and spreads out in one or more of the offshoots of the solar plexus. In a specimen of *Paradoxurus larvatus* I observed it ending in the cæcum.

In many animals the vagi give off plexuses, which usually come from the solar plexus.

*Vago-sympathetic Plexuses.*—In some animals it is very easy to trace a complete plexus along the whole length of the aorta and ending in the pelvis at one extremity, and connected to the cardiac plexuses at the other.

The components of the cardiac and pulmonary plexuses have already been described, and cardiac ganglia are well marked in several animals.

The solar plexus has one or more ganglia in most animals, but no ganglia are present in the drill and baboon. Offshoots from the plexus accompany the various branches of the abdominal aorta in many animals, and the subordinate plexuses have been named after the vessels which they accompany. In some animals the plexuses which come normally from the solar plexus are given off from the vagus, and the fibres contained in them are too numerous to be accounted for by the communications between the vagus and cœliac ganglia. In this connexion Gaskell's statement that the vagi below the pulmonary branches are composed mainly of non-medullated fibres must be taken into account.

The pelvic sympathetic is built on the same plan in all mammals.

The innervation of the endocrine organs is arranged on the same lines in all mammals. These organs have attracted much attention, but the influence of climate on their functions requires more investigation. Bolk, Keith, and others have shown that the characters of man and the anthropoids have been moulded by variations in their activities; and the characters of the various races of mankind are dependent to a great extent on their functions. It is probable that dietetic and climatic factors are the causes which have operated throughout the ages to determine the external appearances of man and the apes. Dietetic causes have acted from within, and climatic factors have acted from without through the sympathetic and vagus.

A study of the comparative anatomy of the vagus and sympathetic nerves shows that much work has yet to be done by the physiologist. At present his investigations are carried out on a small series of animals. If he goes beyond that series and experiments on animals exhibiting extremes of anatomical specialization he will observe phenomena which will help us to understand how certain diseases are produced in man.

SIR WILLIAM BAYLISS, F.R.S. (London).

Using the name "vagus" in an extended sense as meaning that part of the visceral or autonomic nervous system which is not included in the "sympathetic" outflow from the thoracic and upper lumbar segments, we notice that most viscera and blood-vessels are supplied from both of these sources. There is, unfortunately, no satisfactory general name for the whole of the non-sympathetic visceral system. It is sometimes called "para-sympathetic," which suggests that it is in some way subordinate to the sympathetic proper.

But, physiologically, the important point is that, as might perhaps have been expected, the mode of action of these two different kinds of supply on any particular organ is opposite in nature. Thus, the vasoconstrictor nerves are invariably of sympathetic origin, whereas vasodilators come from other sources. Although the action is opposite, it does not follow that the effect produced by each system is of the same sign in all viscera. The sympathetic supply of the intestine is inhibitory; of the vagus, excitatory. In the sphincters, however, the vagus causes relaxation, the sympathetic, contraction.

The local reactions, reflex or otherwise, brought about in the skin by stimuli are clearly of importance in relation to the subject under discussion. I would first call attention to what have been called "Lovén" reflexes in honour of their discoverer. When the anatomical arrangement of the sensory and the efferent vasomotor nerve fibres is favourable for separate action upon them, it is found that stimulation of the former produces a reflex vasodilatation in the organ from which these sensory fibres come, along with vasoconstrictor reflexes elsewhere. Thus the organ receives the maximal blood supply possible. These are true reflexes, because section of the sensory nerve between the point stimulated and the central nervous system puts an end to them. This is not the case with those peripheral vasodilator effects which I called "antidromic." The sensory fibres of the posterior roots, when separated from the spinal cord by section, produce vasodilatation in the area supplied by these fibres, that area from which they come. They do not degenerate when cut between the cord and the spinal ganglion; but do so when this ganglion is removed or when the nerve is cut peripherally of it. They are therefore indistinguishable from the ordinary sensory fibres. It would seem that they must branch somewhere in the periphery, one branch going to the sense receptor, the other to the blood-vessels. Further evidence was afforded by the work of Ninian Bruce, who investigated the remarkable fact that, after the application of cocaine to the skin, mustard oil no longer produces its characteristic inflammatory action. Bruce showed that this effect of mustard was still present after section of the sensory fibres between the cord and the spinal ganglion, and even after section of the main trunk of the nerve, if tested shortly afterwards. But if the fibres were allowed to degenerate, mustard was inactive, just as after cocaine. The only possible explanation seems to be that of branching fibres, giving rise to an "axon-reflex" in the sense of Langley and Anderson. The nerve impulse from a stimulated receptor passes to the point where the vascular branch is given off and then along this in an efferent direction.

It is a somewhat disputed point as to whether there are "trophic" nerves in the sense of their having a direct influence on the nutrition of cells. On the whole, the balance of evidence indicates that even the blisters of herpes may be indirect effects of the vasodilatation. Careful experiments have failed to detect any difference in the rate of healing of wounds in a denervated limb.

There is undoubtedly much work still to be done with regard to reflex effects on viscera produced by stimulation of particular areas of the skin.

Dr. LEONARD WILLIAMS (London).

Each of us is largely the resultant of the forces of his environment. These forces are principally cosmic forces which are beyond and outside our control. We cannot resist them, we can only react to them. We



can adapt ourselves to them, and it is the degree of our capacity for adaptation which leads to success or failure in the struggle for survival. It is our long-continued efforts at adaptation to these cosmic forces which determine our characteristics. Just as conduct repeated so often as to become habitual determines character, so also adaptation, when it has become habitual, determines characteristics. In order to estimate characteristics, man must be measured in the mass. The races of Northern Europe are said to be fair, phlegmatic, but fierce; the Latin races full of pigment, motion, commotion, and emotion; but, to explain these facts, an appeal must be made to environment, and of environment climate is the principal constituent. Now, the most important element in the differentiation of various climates is the sun and the quality of its rays. These rays determine the climate, and the climate determines the physical and mental characteristics of the inhabitants. Colour, for instance, is determined by the quality of the sun's rays. Pigment is developed in the skin for the important purpose of protecting certain organs from certain deleterious rays of the spectrum. This is an adaptation which has become stereotyped. The nerve-endings in the skin to which Dr. Sonntag has referred obviously come into this category: they are delicate structures and require protection. So most emphatically do the endocrines need protection. This is shown by the fact that certain dermic colorations are found to be associated more or less regularly with certain osseous configurations, especially the facial: and in its turn the facial configuration is found to be associated with certain mental and temperamental qualities and defects. The northern races have fair hair, blue eyes, and a type of civilization which seems to encourage hypocrisy: whereas the Latins have dark hair, brown eyes, and a temperament which is at once graphic and pornographic. You remember the saying that the Englishman loves to conceal his vices, whereas the Frenchman is fond of displaying his. If, in order to study the causes of these fairly constant distinctions you transplant an Anglo-Saxon into a Latin land, or a Latin into a northern land—provided in each case you catch him young enough—you will change not only his outward seeming, but his inner man, so fundamentally as to transform him into an aboriginal of his adopted country. But there is a difference. The pigmented Latin, when transplanted, preserves his pigment: whereas the transplanted northerner immediately proceeds to develop pigment; or, if he fails to do this, he succumbs to some disturbance of endocrine function. That the French have recognized this truth is shown by the fact that their Colonial Office will not accept a blond man for service in their tropical colonies, because it has been found that fair men fail to react to their altered environment, and die: whereas the dark Mediterranean type does so react, and flourishes.

Passing from the general to the particular, we will consider one example of a white race in a coloured man's country. The white man went to North America a long time ago, and he has recruited himself steadily from Europe ever since. The continent which he there inhabits is a red man's country, and we are to suppose that the red pigment in the skin was originally evolved for protective purposes. The white man who has taken over this country has developed certain characteristics which, viewed in the mass, are quite distinctive. If the characteristics are studied, it will be observed that they are the result of over-stimulation of the endocrines, and of one endocrine in particular, the pituitary. The pictures of "Uncle Sam," as usually drawn, represent a tall, lean man, with a tendency towards giantism, with well-developed maxilla and mandible, and with plenty of dark straight hair which



he shows no tendency to lose; this is quite different from the ordinary Anglo-Saxon or Scandinavian type from which he is supposed to be derived. Now, if we consider the matter, we are driven to the conclusion that this change is due to over-stimulation of the pituitary. In order to protect that gland from the rays of the solar spectrum which originally caused the evolution of the red colour in the aboriginal, the hair of the head of a born American is abundant and dark, and he seldom or never becomes bald. But the protection afforded by the hair is never quite sufficient, for if you look at him carefully, our typical American shows signs of what we should call commencing acromegaly. The picture shown taken from Cushing's book exemplifies this.

My friend, Dr. Woods Hutchinson (nephew of the late Sir Jonathan Hutchinson), tells me that he went to America when he was aged about 13. He was then a red-headed, fair-skinned, typical English boy. Residence in the States turned his hair black, and developed a facial type which is unmistakably American.

This question of the purpose of hair in protecting certain endocrines is interesting. The pubic hair might easily be endowed with such a function, and it is significant to note that the distribution of this hair differs in the two sexes. In the female it is much wider, as though to cover the ovaries. The beard in man may be designed to protect the thyroid, this gland in the male being much less vigorous than in the female. These statements, however, are merely suggestions.

Lastly: The question of dermic pigment is most important from the imperial standpoint. The English are the great colonizers, and if it be true, as I feel sure it is, that the white man can survive in the coloured man's countries only on the condition of his being able to produce the necessary protective pigment, then our Government should take a lesson from the French and discourage fair people from emigrating to such countries.

DR. F. HERNAMAN-JOHNSON (London).

The addresses on the anatomy and physiology of the vagus and sympathetic to which we have listened should teach us to beware of hastily formulating theories as to the precise action of remedies which produce their effects through the nervous system.

Electrical treatment is frequently employed by those who specialize in hydrology, so that some remarks as to the clinical effects of certain electrical modalities in abdominal disorder may be appropriate.

When I began to "read up" medical electricity out of text-books some fifteen years ago, it was laid down that interrupted currents should never be used when there was spasm, as they made it worse. This may perhaps be true as regards voluntary muscles, but as to spasm of the stomach and bowels which is not the result of a definite organic lesion, it is not in accord with clinical experience. Rhythmical stimulation of the muscles at the front and sides of the abdomen has an excellent effect upon spasm. This is proved by the gradual disappearance of the pains which result from a portion of bowel attempting to force its contents through a spasmodically contracted section; and it can also be directly shown by X-ray examination before and after a course of such treatment.

It used to be taught that such rhythmical stimulation of the abdominal muscles could only be of benefit when the muscles are flabby. In many cases

which have come under my observation, however, the muscles were firm and apparently not in need of strengthening. Nevertheless, stimulation of them got rid of any bad symptoms. Apparently we must fall back upon the general explanation that interrupted currents "restore tone" to the vagus and sympathetic nerves, and tend to promote their normal function. While this statement is vague, it is at least not dogmatic, and has the advantage that it encourages us to try similar currents in other cases of disturbance of the sympathetic, e.g., Graves' disease.

Sir William Bayliss has described how the usual effects of oil of mustard on the skin may be prevented by paralysing certain nerve terminals in the skin. It is of interest in this connexion to recall certain experiments made at Haslar during the war, in which the blistering effects of a glowing cigarette end were prevented by the suggestion, under deep hypnotism in a sensitive subject, that there should be no pain. In fact, one of the peculiarities of the sympathetic nervous system—and possibly also of the extended vagus—is that the same effect may often be produced either by a physical or psychical cause. Graves' disease may be brought on by physical exhaustion or emotional stress—an attack of asthma by the unknown presence of a horse, or by the unjustified belief that a horse is near. It should therefore be pointed out that whenever disorders such as those just mentioned have to be treated, the mental side of the case should never be ignored.

Dr. L. SCHMIDT (Pistany, Czecho-Slovakia).

My remarks will be limited to discussion of the relation of hyperthermal mud baths to the sympathetic nerves. In his treatise on "Principles and Practice of Medical Hydrology," Fortescue Fox says:—

"The skin is a nervous organ for relationship with the external world. This nervous surface is constantly engaged in receiving and transmitting to the centres in the brain and spinal cord an infinite number of impressions from the outside world."

The truth of this statement will be recognized by all medical practitioners engaged in the practice and study of balneology, when we come to consider the fact that obviously all balneary treatment starts with an attack upon the skin. It is the skin that represents the great battlefield of Catalaunum, on which the balneo-climatotherapist is to settle the fight between his therapy and the enemies of the organism.

The outstanding factor in treatment with hyperthermal mud is, as in all similar procedures, the marked hyperæmia set up by the action of the sympathetic nerve system of the skin.

The sensational researches and discoveries of Bier and Dastre-Morat attribute absolute curative properties to this condition of hyperæmia, but in reality they fail to establish the exact reasons for the result. Winternitz's explanation of the incitement to increased activity of the natural defensive and protective apparatus of the organism, also the idea of Goldscheider, who considers the increase of the resistance against infection as a great collective action, are equally of the nature of generalizations.

In the course of our balneo-clinical observations at the Industrial Hospital of the Spa at Pistany, my colleague, Dr. E. Weisz, and myself came to the conclusion that all these explanations offer an excellent basis for further clinical research, but far too often fail in giving sufficient guidance in practical

therapeutics. Consequently we began to make further fundamental researches. Encouraged by the doctrines of the laws of immunity, more especially of those established by Wright, and in consequence of years of experience, we have today arrived at the point of considering the effect of baths mainly as one of a protein and vaccine therapy. We maintain that all we do in hyperthermy treatment is to transmit in large quantities into the blood-stream, from the exudation depots, either matter entirely foreign to the body, or, if a property of the body, yet foreign to the blood, according to the nature of the disease. This matter then sets up either a specific or a general immunizing activity, by calling into action the bio-chemical defensive properties in the blood and greatly strengthening the vitality of the protoplasm.

Consequently we do not share the view of Bier and Dastre-Morat that hyperemia itself constitutes the actual curative factor, *in loco morbi*, but rather that it takes the place of a vehicle for proteins and vaccines, and hence we may compare it to the hypodermic needle, by the aid of which albuminoids are usually administered. Hence we see that the vasodilator endings of the sympathetic nerves play the part of door-keepers that control the entrance of the reactionary and, at the same time, curative agents.

But the part played by the sympathetic nerves during treatment with hyperthermal mud does not end here. Certain chemical components of the mud that directly stimulate the (sympathetic) nerve-ending, seem equally to influence certain endocrine activities which give rise to the protective and defensive bodies of the organism.

The validity of these statements is established by the following observations: In certain cases of obstinate chronic rheumatoid arthritis, we are in the habit of laying much prognostic stress upon the presence of minimum degrees of febrile reaction. We usually abstain from thermal treatment in such cases, until we have succeeded in entirely abolishing these rises of temperature by other therapeutic measures. For this purpose we sometimes recommend energetic friction all over the body with alcohol, but it should be pointed out that in so doing, we recommend entire avoidance of the affected joints, because our primary object is to prevent increased outflow of proteins into the blood-stream. There can, therefore, be no question of any direct hyperemic influence at the seat of the disease, and yet we occasionally find that the ominous minimum rises of temperature vanish after a relatively short space of time.

The cause for this improvement resides in the fact that the mechanical and chemical stimulus of the alcoholic friction upon the nerve-endings, as above mentioned, increases the activity of internal secretions in the defensive apparatus.

We are supported in our conviction by the profound researches of the Italian school, especially those made by Pende, and by the important work of Langdon Brown, all of which emphasizes the intimate relationship between the autonomic system and the endocrine functions. In the same way we confirm the accurate investigations made by von Furth, who succeeded in proving that endocrine organs the functions of which are actuated by the sympathetic exercise an accelerating influence upon metabolism, stimulating protein destruction, carbohydrate mobilization, and also the metabolism of fats as well, while they also control the water and salt output and the galvanic irritability of the nerves.

This also explains why it is impossible to get the same therapeutic results

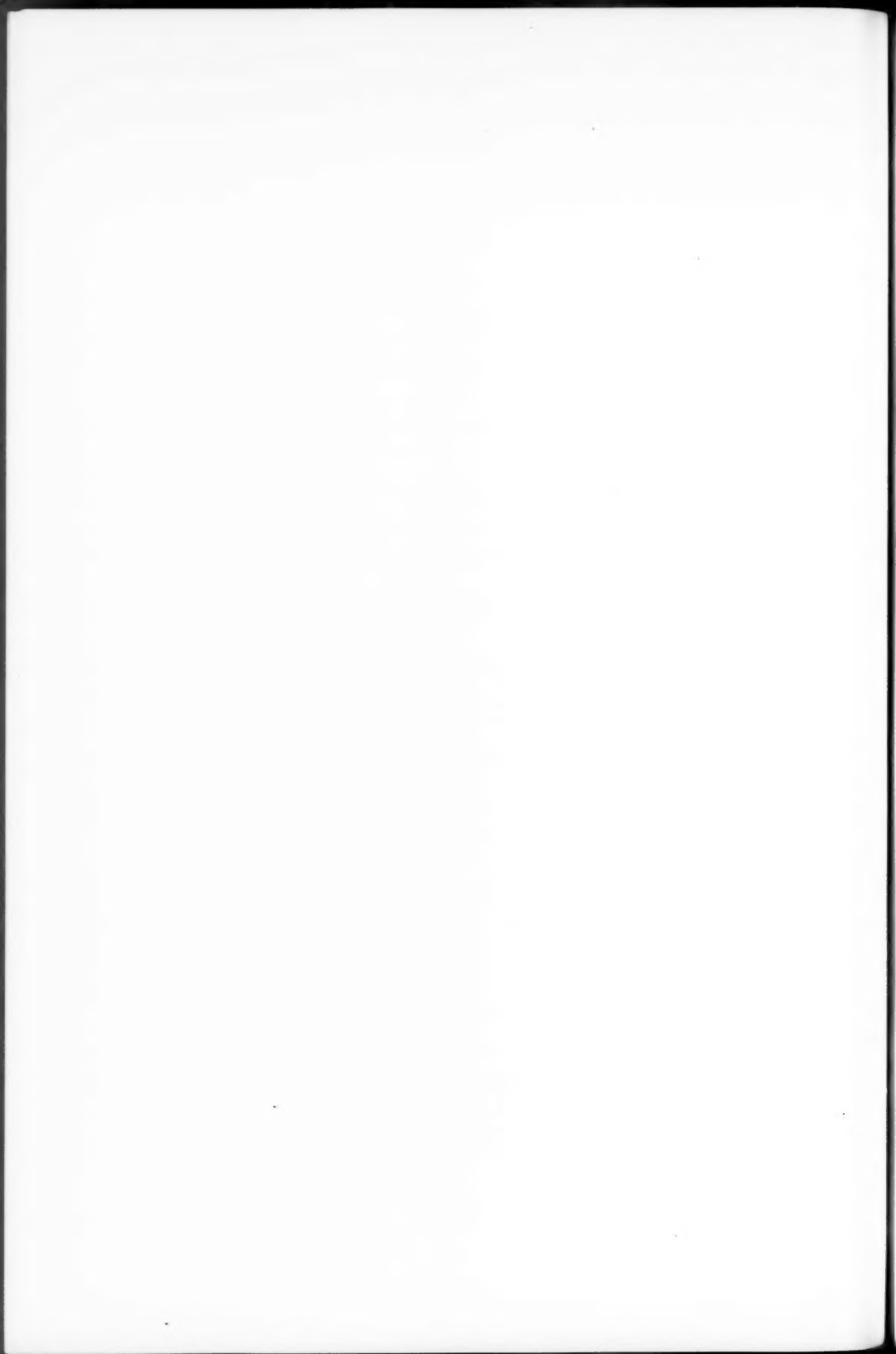
from the mere application of warmth, that we get from treatment with thermal mud; the silicic acid, sulphur, and radium contents of which have so beneficial a chemical reaction on the nerve-endings in the skin, together with the influence of warmth.

The PRESIDENT (Dr. Campbell McClure).

The object of this discussion on the vagus and sympathetic is to try to turn the minds of those who are engaged in spa practice towards the basal facts underlying the effect on internal organs of applications to the skin; to enable such men to give a reason for the clinical faith that is in them; to convince the general physician that spa methods have a definite effect in the treatment and cure of disease and are not merely a camouflage for treatment by rest and diet. It would seem that on the maintenance and restoration of the normal balance between the vagus and sympathetic depends in a considerable measure the general health of the patient, and any form of treatment which has as its result such maintenance or restoration is of the greatest possible value. Enough has emerged from the discussion to show how intimate are the connexions between the skin and the circulation, between the skin and the abdominal viscera, and how easily the state of the circulation and of these viscera can be influenced by applications, soothing or stimulating, to the skin.

One important detail is worth emphasizing—the necessity for calculating very carefully the temperature of all general applications to the skin, regard being had to the fact that vagus asthenics are intolerant of hot baths while those in whom the vagus system is over-active are equally intolerant of cold baths.

One dares to hope that a discussion such as this may stimulate to further activity the scientific curiosity of all those using hydrology as a serious method of treatment with the result that the therapeutic employment of waters may have an application, if not more general, at least more particular and accurate than is the rule at present.



## Section of Balneology and Climatology.

President—Dr. J. CAMPBELL McCLURE.

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### The "Aërologia" of Domenico Panarolo.<sup>1</sup>

By GUY HINSDALE, M.D., Hot Springs, Virginia.

THIS little volume, published in Rome in 1642, is by Domenico Panarolo, designated on the title page as a Roman philosopher and physician. It is dedicated to Signor Fabritio Naro and to "the illustrious guardians of the Hospital of the Holy Saviour," at which the author practised medicine. Panarolo was versed in the current knowledge of animals, vegetables and minerals, together with pharmacy and last, but not least in his estimation, astrology.

Domenico Panarolo was born in Rome in 1587, and died in the same place in 1657. He studied medicine and surgery in the Laterano Hospital under Pierre Castelli. He dedicated himself to the study of botanic science, which, by order of Pope Innocent X, he taught in the University of Rome, where, later, he taught anatomy. He was director of the botanical gardens of the Semplici in Rome and Messina and was chief physician of the Laterano hospital. He was a philosopher, a naturalist, a botanist, an anatomist, a hygienist and a meteorologist ranking above the majority of his contemporaries and he was an honour to the field of medicine. His style in writing was elegant and often poetical. He undertook scientific travels through France and Germany, and the largest academies strove to count him among their members.

The volume bears what is apparently a library imprint; this consists of a Sacred Heart with three stars encircled by the words "EX CONGR. S. PHILIPPI NERII BONONIAE" and it is interesting to note that on March 12 of that year (1642) there was celebrated in Rome the three-hundredth anniversary of the canonization of St. Philip Neri, the founder of the Congregation of the Oratory.

After an opening chapter on the nobility of man the author in the second chapter discourses on the importance of health, agreeing with Plato: "Primum sanitatem; secundo pulchritudinem; tertio vires ad cursus et alios corporis motus; quarto divitias." Health is first and riches last. From St. Augustine's writings he quotes that "it is better to have the stature of Zacchæus with health, than to be a Goliath with fever."

The third and fourth chapters treat of the air and its importance for the human race, and contain a description of the movement of the blood, which is

Panarolo (Domenico): *Aerologia*, cioè Discorsa dell'aria, trattato utile per la Sanità.—Roma, 1642.

not quite clearly explained, though this book was written fourteen years after William Harvey's "*De Motu Cordis et Sanguinis in Animalibus*," which, by the way, Panarolo does not mention: "It is warm and moist in its nature, as we have said, but in comparison with our natural heat it is cold; wherefore it happens that the heart attracts it to itself through the lungs by means of the venous artery (*arteria venosa*) and the left auricle, ventilating its heat in diastole, or in that movement when the heart is dilated, sending a sufficient portion to the great artery in order to moderate the universal heat of the body and because in the heart, as in other material fire, a large amount of steam is generated continually with a great abundance of vapors and smoke which, should it happen to get closed, would extinguish the native heat not otherwise than in case of flame, which not being able to have fresh air is extinguished in the same smoky exhalation (*esalatione caliginose*) just as the heart by means of the above mentioned air throws it out in systole, or in the movement when it contracts in the same way by which it had attracted it, which is clearly shown in anatomy, of which I have made a particular study."

Fire was considered for two thousand years the source or agent of motion, and fire and the heat of it in all things. This theory of innate heat, *ἐμφυτον θερμὸν*, constituted a dogma which governed physiology down to the day of Thomas Willis (1621-1675). It is a very fascinating subject, on which Sir Clifford Allbutt has written a scholarly article.<sup>1</sup>

Fire and air were held to be close allies or, as Heraclitus affirms, fire was "the finest and subtlest air." This animating fire was not visible flame; it was something subtler, something between air and fire, penetrating and vitalizing everything. Respiration was then regarded as the chief path by which this fiery air entered and penetrated the body; the blood made in the liver generated heat which was carried to the heart, the heat centre of the animal system.

With reference to the Aristotelian dogma of the innate heat Huxley, in his Harvey Lecture, summarizes Galen's view as follows: "Part of the blood, it is supposed, went through what we now call the pulmonary arteries and branching out there, gave exit to certain fuliginous products, and at the same time took in air from a something which Galen calls the 'pneuma.'" Sir Clifford Allbutt says: "Harvey . . . relied, as we have seen, on the innate heat, and in the derivation of the motion of the heart from the celestial essence; so he likewise regarded the lungs as cooling fans."

Conforming to the custom of those days 118 authors are quoted, ranging from St. Augustine to Virgil. There are interesting and appropriate quotations from the Bible, Hippocrates, Ovid, Horace and Avicenna.

In the fifth chapter Panarolo plainly states his belief in astrology, quoting Tasso as follows: "Viewing causes and effects and the motions of the celestial bodies and the planets, rain, thunder, the breath of the south wind (*notus*) and when the sea is rough and when it rains."

The celestial bodies are the cause of all changes and every movement in this lower world. "The science of the stars is the groundwork of medicine."<sup>2</sup> "A physician without astrology is as an eye which has no power to act."<sup>3</sup> "It is necessary for a physician to know and have confidence in the nature of

<sup>1</sup> "Contributions to Medical and Biological Research" (dedicated to Sir William Osler). New York: 1919.

<sup>2</sup> "Medicus sine stellis et necromanta sine ossibus mortuorum est quasi imago quae non est a spiritibus adjuta."

<sup>3</sup> "Medicus sine astrologia est quasi oculus qui non est in potentia ad operationem."



the stars and their operations so that he may have knowledge of different diseases and of critical days, for nature herself is in accord with the appearance and recognition of the heavenly bodies."

Panarolo says that: "Copernicus asserting the movement of the earth (an opinion repugnant to sacred Scripture and the Holy Roman Church) holds that the wind has its origin in matter."

In the sixth chapter sixteen winds are enumerated as follows, from north to north-west, and so around the circle:

- (N) Boreas, Circio, Coro, Iapige;
- (W) Zefiro, Argeste, Africo, Africo noto;
- (S) Noto, Leuco noto, Euro, Vulturno;
- (E) Apeliote, Ceciapeliote, Cecia, Aquilone.

All these winds and their sources are described, their influence for good and ill is fully discussed, and there are many notes on the derivation of the various names. For instance, Zephyr is the *Ζεφύρος* or life bearer; Notus, also known as Auster, and Fenicia or Leuconotus seem to have had a particularly bad reputation; Apeliotes or Subsolanus (the east wind) had an excellent reputation as being cold, dry, opposed to all contagion, and a preserver of the body and of sanitation.

He considers that Ceciapeliote (east-north-east as we should say) was responsible for quotidian and tertian fever by reason of its warmth, particularly in summer.

He also notes that winds in passing over snow fields, marshes, swamps, lakes, seas, dead bodies, &c., may degenerate or vary from their true character.

The seventh and eighth chapters are on climates and their number, which is seven. These are defined strictly according to latitude, the length of the longest day and the height of the pole star, which is given in degrees and seconds together with geographical boundaries. In the seventh climate he includes the island of Hibernia, called Irlanda; Albion or Inghilterra; Scotia; and the Orchades (Orkneys).

The ninth chapter is on the choice of a habitation and its exposure to the air.—Persons living in the higher habitable places are healthy and strong, able to endure much labour and are long lived.<sup>1</sup>

The last chapter is on the contamination of air. He relates cases of suffocation from burning charcoal; from the fumes of Vesuvius and from breathing the air coming from the burial places in certain churches. The air of certain caves, grottoes, and subterranean places is especially dangerous and fatal.<sup>2</sup>

<sup>1</sup> In locis habitabilibus altis morantes sunt sani et fortes, laboris multum patientes et vivunt diu."—AVICENNA.

<sup>2</sup> Thanks are due to the librarian of the Boston Public Library, the New York Public Library and the College of Physicians of Philadelphia, for notes regarding Panarolo and his published works, and to Rev. Dr. A. B. Hunter, of Florence, for the volume here noticed.

## Section of Balneology and Climatology.

President—Dr. J. CAMPBELL McCLURE.

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### The Use of the Plombières Douche.

By ALEXANDER GEORGE GIBSON, M.D., F.R.C.P.

BY the employment of this method on an extended series of cases in which there were abdominal symptoms I have been convinced of its great value, as it means both of diagnosis and of treatment.

The particular method adopted is one which can be carried out by any intelligent nurse, and is as follows: A preliminary soap-and-water enema is given in order to obtain a complete evacuation of the colon. This is an essential feature and if adequate evacuation is not obtained the enema may have to be repeated, with or without the addition of turpentine or glycerine. The result of the enema is carefully noted and then the douche, warmed to the body temperature, is administered. This consists of a 0.7 per cent. saline solution to which may be added any antiseptic or drug which it is desired to apply to the surface of the colon. It is given by a rubber catheter and rubber tube (from 18 to 24 in. long) with a funnel attached. The catheter need not be inserted more than 2 or 3 in. The patient first lies on his left side, and the saline is slowly poured in. After two minutes the position of the patient is changed and he lies on his back: and after another interval of two minutes he lies on his right side; this change of position facilitates the flow of saline along the different parts of the colon. The nurse is instructed to report any symptoms of pain or discomfort complained of by the patient and whether there is any stoppage to an even flow. For the first douche I usually direct that not more than one pint should be injected; subsequent douches may be increased up to two pints. The douche of saline is usually quickly returned and in the first part, mucus, pus or flakes of mucous membrane (uncoloured, or coloured by faeces) and sometimes concretions, appear. The presence of mucus or mucous membrane indicates surface abnormality, and any such discharge should be saved for naked eye and microscopical inspection. Very valuable information may be obtained occasionally by a careful microscopic examination of the product both unstained and stained, and, whenever possible, it should be sent for such examination. Parasites, pathogenic amœbæ and bacteria, are found here less contaminated by other faecal bacteria, and the well-washed flakes of mucus form the only practical means of obtaining from the colon cultures likely to be uncontaminated.

The method is as valuable for diagnosis as for treatment, and it is in its routine application in clinical examination that its results have been so valuable. The presence of mucus or membrane in the returned saline indicates catarrh or necrosis in the mucous membrane or a portion of it, and a painful

[May 5, 1923.]

spot may enable such abnormality to be accurately located. A careful inquiry about the patient's symptoms after the douche is important. Sometimes there is a sense of comfort, in which case we may presume that the lavage has done good; in other cases the patients complain of more discomfort, and when it may be that some inflamed area has been stirred up. The source of the catarrh is not necessarily in the bowel itself: it may be in the peritoneum or in some neighbouring organ.

As a therapeutic measure I have used it in the following conditions: (1) Colitis of all types especially that following appendicitis. (2) Sciatica and myalgias of the lumbar region in which a coexistent mucous catarrh is frequently found; in one of the cases observed the result was most striking. (3) Pyelitis and bacilluria when methods of diet, purgation, and antiseptics have failed to bring any amelioration of symptoms. (4) Gastric and duodenal ulcers; the method often reveals unsuspected abnormality such as old scybala and mucus whose presence would delay recovery. (5) Certain cases of rheumatism and arthritis showing colonic symptoms; treatment by this method improves the general condition of the patient and so puts him in a better state to battle with the disability of the joints.

It is not claimed that by the removal—temporary or permanent—of colonic catarrh the patient is cured, but when the source of the catarrh lies in the bowel, the douche, daily or less frequently given, makes a considerable difference in the symptoms. As a means of ascertaining the state of the internal lining of the colon it gives results which are valuable both in conjunction with and independent of the clinical evidence.

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SECTION FOR THE STUDY OF DISEASE IN CHILDREN.



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## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

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### The Pathogenesis of Rickets:

#### PRESIDENT'S ADDRESS.

By ERIC PRITCHARD, M.D.

I MAKE no excuse for selecting rickets as the subject of my presidential address, because in the first place I have given more attention to this disease than to any other, and secondly because I regard its various manifestations and fore-stages as the most important and widely spread pathological symptoms of childhood. If we could abolish rickets by any of the various panaceas which have been recently suggested, I submit that we should at the same time necessarily abolish all those minor forms of malnutrition which are antecedent to it.

It has been suggested to me that this is not exactly the psychological moment to attempt to resuscitate the theory of the causation of rickets which I originally explained before the British Medical Association's annual meeting at Cheltenham in 1901, for half the world at the present moment, owing to the somewhat premature and sensational claims of certain experimentalists, appears to believe that the key to rickets has already been discovered in the fat-soluble "A" factor, while the other half apparently believes that sunlight or the vapours of a mercurial lamp can cure in a few minutes all that has been accomplished by bad feeding and bad hygienic conditions in as many months. For my part I believe it is the proper time and the proper occasion to utter the strongest possible protest against the making of premature deductions on so important a matter from a few laboratory or clinical experiments. It is universal knowledge that you can, with almost complete certainty, produce rickets in children or animals by any flagrant contravention of the laws of hygiene, and that you can cure, or at least improve the condition by any measure which removes or mitigates the particular hygienic factor at fault; but this does not prove that the particular offence in question is the sole cause or the *causa causans* of rickets, it simply proves that under the existing circumstances it has acted as a determining factor.

If I do not draw exactly the same deductions from the great mass of experimental evidence that has accumulated of recent years on the subject of the causation of rickets as are drawn by the authors of the experiments themselves, I yield to none in my admiration of the value of the many splendid contributions to our knowledge of the precise effect of various environmental factors on the metabolic processes. I welcome each of these contributions, not only as pieces of exact evidence but also as essential elements in the fabric of my own theory without which the latter would fall to the ground.

The great majority of these recent researches on the influence of vitamins, phosphorus, calcium, sunlight and infective organisms teach us nothing new nor do they in any way throw light on the essential morbid process, but they do place on the sure foundation of experimental proof much that we had believed or suspected on clinical grounds. There is nothing new in the belief that want of sunshine is the cause of rickets; we have believed this, and have acted upon it in our treatment from the time of Glisson downwards. There is nothing new in the suggestion that rickets can be caused from want of fat; I was taught that by my old teacher, Dr. W. B. Cheadle, in the year 1889. There is nothing new in the vitamin theory except the name and the classification; moreover a great deal of what we have recently been taught by the pundits of this school we shall have to re-learn.

If rickets can ultimately result, as most of us know from experience that it can, from deprivation of sunlight, bad feeding, want of exercise, constant exposure to infection, and all the other concomitants of slum life, how can we reconcile such a belief with the knowledge that exactly the same result can accrue in the palace where probably none of these factors are in operation? These anomalies and seeming contradictions can only be reconciled by predicating some central *causa vera* to which all these predisposing factors are contributory.

I suppose there is no problem in the whole domain of medicine which has received so much attention at the hands of our profession as the aetiology of rickets; there is scarcely a physician of repute who has not, at some time or other attempted to sever the Gordian knot and explain the causation of this elusive disorder on the ground of some one particular fault of diet or hygiene. At the present moment most investigators of rickets are ranging themselves in one or other of two camps; while one regards the disease as a dietetic disorder, the other refers it to some environmental cause or causes. In the first place it seems to me to be illogical to draw a distinction between a dietetic and an environmental disorder: surely diet is as much a part of the environment as sunlight or exercise? And in the second place there seems to me to be nothing mutually exclusive in the two hypotheses; on the contrary, I feel convinced that the two are mutually inclusive and necessarily complementary. The deeper each party probes into the problem from its own particular point of view, the nearer it seems to approach to the views of the other, but neither of them has yet reached the pith or kernel of the problem, or has explained the *modus operandi* of the essential pathogenesis. The theory which I venture to submit in reconciliation of both hypotheses is as follows: All conditions of malnutrition in infants and young children arising from any cause whatsoever, whether of bad-feeding or mal-hygiene, provided they are severe enough and long enough continued, inevitably lead to the final condition we call rickets for the reason that all such conditions conduce to the development of an acidosis which itself entails that mineral depletion and calcium impoverishment which are the pathognomonic evidences of the true rickety condition.

Before proceeding further with the argument on which the acidosis theory is based, it may be desirable to state exactly what I mean by the term "acidosis," for unfortunately this much abused word is subject to very varied interpretations. In the sense in which I use the term I do not mean the presence of acetone in the urine, in the blood, or in the breath, nor do I mean a shifting of the acid-alkaline balance of the blood in the direction of acidity, although each and all of these complications are common, and at times obligatory, results of an acidosis. What I do mean is an excessive production

of acid bodies other than  $\text{CO}_2$ , and the loss of alkaline reserves which is entailed by their neutralization. In a compensated acidosis such as usually exists, at least in the early stages of rickets, it is the compensation itself, i.e., the sacrifice of the alkaline reserves of the blood which leads to the familiar symptoms.

A great many experiments have been made by various investigators to gauge the degree of acidosis or of mineral depletion which exists in different pathological conditions. It cannot, of course, be estimated by the hydrogen-ion concentration of the blood; it can only be accurately estimated by measuring the actual amount of carbonates in the blood and in the reserve depots of the bones and other tissues. Even in extreme conditions of mineral depletion in which the alkaline reserves of the blood have been reduced to a dangerous level, the amount of calcium in the blood need not be seriously reduced. All forms of calcium are not available for the neutralization of acid bodies, nor are all forms used for neutralization purposes. For instance, phosphate of calcium is of no value for this purpose, nor is the chloride. Hence the amount of calcium in the blood at any moment is no index of the amount of ionic calcium available for neutralization purposes or for tissue growth. Moreover, as a valuable and expensive base calcium will not be used for neutralization purposes until the supplies of the cheaper bases, ammonium, sodium, and potassium, have been exhausted. Nevertheless, by more accurate and delicate methods Stheeman [1] has been able to show that during active rickets there is practically always some falling off in the amount of calcium even in the blood, while others [2] have attempted to show—a much more difficult matter—that there is a falling off in the amount of the “available” calcium in the blood.

If in conditions of acidosis there is an urgent claim for alkalis and available metallic bases to neutralize acid bodies and thus prevent the shifting of the acid-alkaline balance to the acid side, it is clear that there will be a smaller residuum of these elements for the far less urgent purpose of mineralizing bone. It must be remembered that bone cannot be mineralized with phosphate of calcium alone. The latter is a comparatively cheap salt in the animal economy, but the equally essential calcium carbonate may, under conditions of acidosis, be very precious and very difficult to obtain. The defective mineralization of bone such as occurs in rickets is, therefore, on theoretical grounds to be expected in mild conditions of chronic acidosis; while in cases of severe need calcium carbonate may even be extracted from available depots in normally ossified bone, as indeed takes place in osteomalacia.

Dr. E. P. Poulton [3] somewhat caustically remarks: “It has been gravely suggested that the acidosis in rickets dissolves the lime salts from the bones—an obviously impossible result without a rise in the hydrogen-ion concentration in the tissue fluids surrounding the bone.” One might equally well discredit the possibility of the chick developing *in ovo* being able to draw upon the calcium in the shell for the calcification of its bones. Moreover, if the views of Ylppö [4] and Bengt Hamilton [5] are well founded, the full-time baby invariably draws upon depots of calcium in the bones to make good the natural deficiency in milk. This fact, if it can be substantiated, may well explain the well-recognized absence of rickets in young babies, and its comparative frequency in premature babies in whom there are no reserves. As I shall again emphasize, it must be remembered that all forms of calcium are not equally available for the purposes of metabolism. Calcium phosphate, for instance, is not available for neutralizing acid bodies.

In the infant the bones can wait for their calcium without jeopardizing life, but the blood cannot wait, for its presence in this fluid is obligatory to fulfil certain vital metabolic functions. Hence in conditions of chronic acidosis in the developing child life is saved at the expense of the bones, and hence the defective mineralization of bone and the presence of other symptoms due to mineral depletion.

Since the whole question of acidosis or the production of an excessive amount of acid bodies other than  $\text{CO}_2$  must necessarily depend on some defective metabolic capacity to oxidize food elements to their normal end products  $\text{CO}_2$ ,  $\text{H}_2\text{O}$  and urea, and since failure to do so must depend either

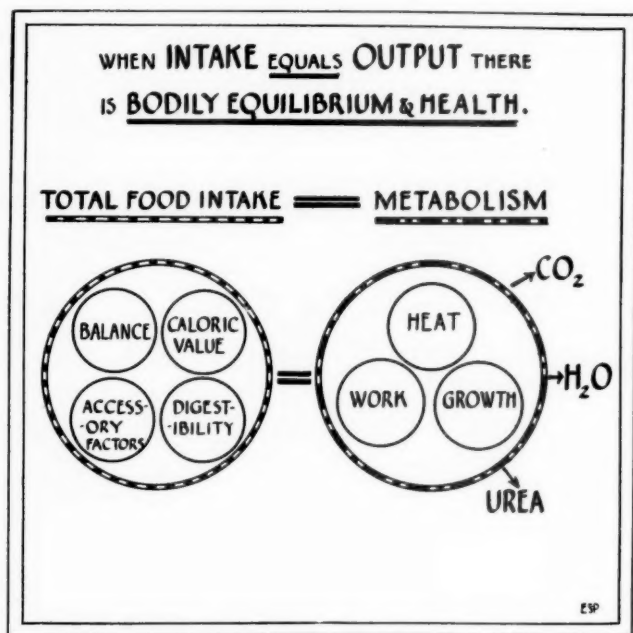


FIG. 1.

on an excessive supply of food or on some defective power of oxidation, I shall now proceed to discuss the conditions on which incomplete oxidation depend.

In conditions of organic equilibrium or health there must be more or less correspondence between the value of the potential energy of the food consumed and the physiological demand for it, i.e., for the purposes of growth, heat production, kinetic energy and secretion. If the two are not equated there must be want of organic equilibrium or ill-health. If there is an excess of intake over physiological output the superfluity of potential energy may be disposed of either by a wasteful or pathological increase in the amount of heat produced, or, if for any reason this method of disposal cannot be resorted to,



there will be a restriction of oxidation with the production of a corresponding quantity of semi-oxidized products; in other words, there will be an increased output of acid bodies of large molecular size, which must be neutralized. On the other hand, if the intake, as compared with the physiological output, shows a deficit, in order that the two sides of the equation may be made to correspond, there must be some restriction of growth, in the output of heat or in the display of energy. If all the potential energy of the food consumed is employed for physiological purposes, it is quite clear that there can remain no surplus energy to leave the body in the form of semi-oxidized end-products, consequently all food elements will leave the body in the completely oxidized

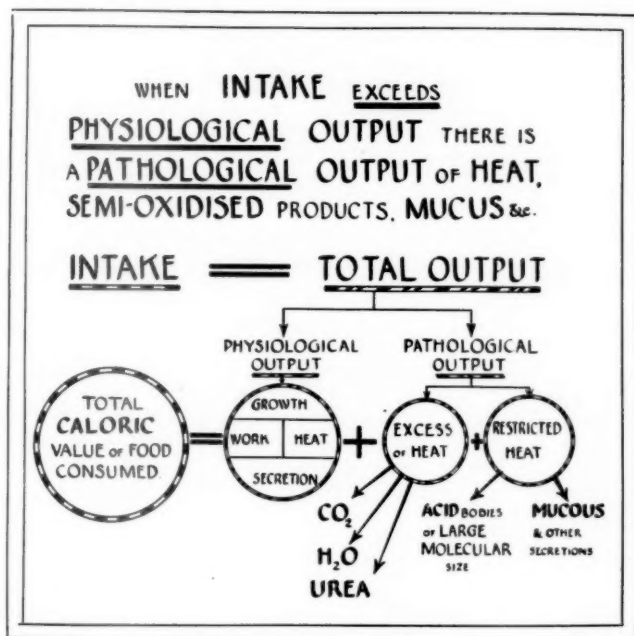


FIG. 2.

and energy-free forms of  $\text{CO}_2$ ,  $\text{H}_2\text{O}$  and urea. On the other hand, if there is a surplus of energy left over after all the physiological functions of the body have been performed, the excess must be disposed of by a pathological output of heat, or by an increased output of albumin, sugar, mucus, or some other unoxidized or semi-oxidized material.

Fig. 1 shows diagrammatically the condition of health or bodily equilibrium in which the calorie value of the food intake is equal to the physiological output, and in which the products of metabolism are represented by the completely oxidized and energy-free bodies  $\text{CO}_2$ ,  $\text{H}_2\text{O}$  and urea.

Fig. 2 exemplifies a condition of unstable equilibrium in which the caloric value of the intake is greater than the physiological output of energy. In this case the surplus energy must be disposed of pathologically in the form of an excessive heat production or by an excessive output of semi-oxidized bodies or acids of large molecular size. Which of these two alternatives will be adopted in any particular case will depend on concomitant circumstances, chief among which are the amount of accessible oxygen, and the available opportunities for heat dissipation. As a rule both expedients are called into requisition, but in accordance with the law of physiological expediency the share taken by each will be that which, under existing circum-

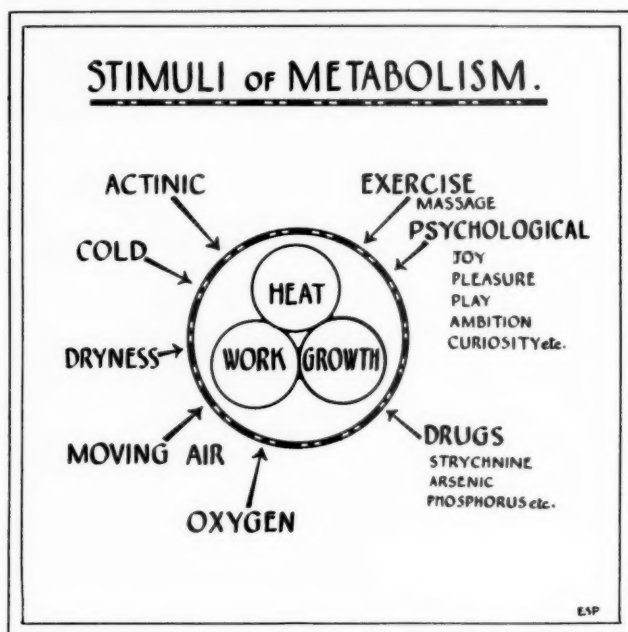


FIG. 3.

stances, will inflict least damage on the organism as a whole. Excessive heat production has at times pathological penalties which are no less serious than restriction of oxidation with a production of semi-oxidized bodies; moreover, there may be conditions—as in anæmia for instance—in which the supply of oxygen is limited, and in which complete combustion is impossible.

It is clear that in conditions of ill-health in which the output of energy is less than the total value of the potential energy of the food, physiological output can be equated to the intake and health restored by any expedients which can increase metabolism to the required degree, as for instance by the taking of increased exercise, or by exposure to increased cold, as shown diagrammatically in fig. 3.

It will be noticed that the stimuli of metabolism indicated in the above diagram are exactly the expedients most successfully employed in the preventive or curative treatment of rickets.

Any existing condition of equilibrium or bodily health in which the physiological output is equated to the energy value of the food intake can be disturbed by all events which depress metabolism or restrict the output of energy. Such depressants of metabolism include a high external temperature, stuffy atmosphere, redundant clothing, limitation of exercise, want of sun, mental depression and so on, as shown in the accompanying diagram (fig. 4).

It will be noticed in this schema that the depressants of metabolism include most of the well-recognized ætiological factors which singly, or in com-

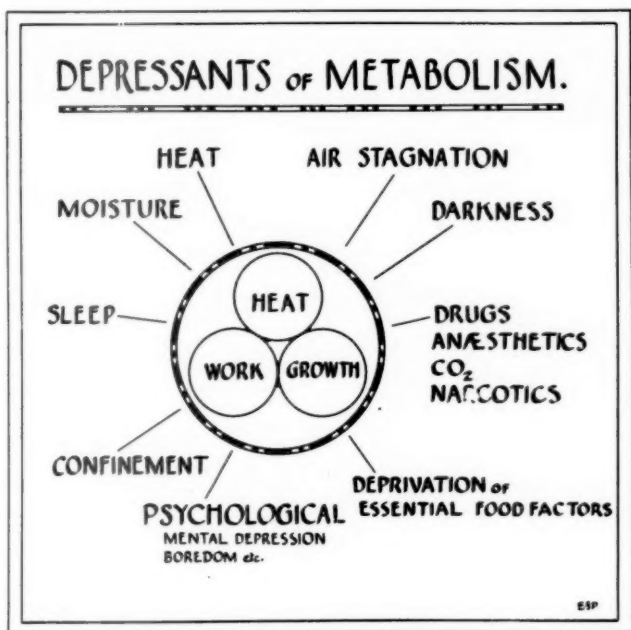


FIG. 4.

ination, predispose to rickets by interfering with the efficiency of the body as a working machine, and thereby limit the output of physiological work.

It will be gathered from the foregoing explanations that an excessive production of semi-oxidized products of metabolism is the inevitable result of any circumstances which interfere with the working efficiency of the body as a whole, and further that the same result follows from an excessive intake of potential energy in the food. In either case part of the energy must be side-tracked into pathological channels in accordance with the laws of the conservation of energy, and this is, I submit, the reason why both the dietetic and the "so-called" environmental theory of the causation of rickets are complementary to one another.

A diet which is essentially rachitic can be rendered physiologically correct if, under the existing circumstances, it is possible by appropriate forms of stimulation to make the child display a corresponding degree of energy. On the other hand, an environment which is well designed to produce rickets

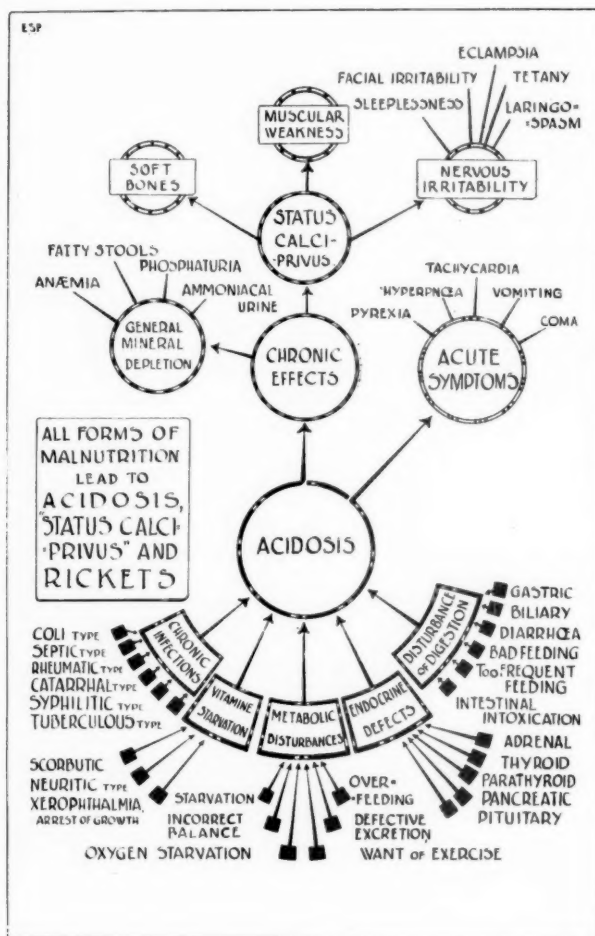


FIG. 5.

may be shorn of its immediate dangers if the intake of food can be adjusted to the existing capacity of output. Whether, in any given case of rickets, we are justified in saying that the remote cause is a faulty environment or a faulty diet, depends very largely on how we look at the question. Neither view

is right, and yet neither view is quite incorrect. It would perhaps be more logical to say that the cause, that is to say the *causa vera*, is a faulty metabolism contributed to both by a faulty diet and by a faulty environment.

In the great majority of cases of rickets the *ensemble* of aetiological factors which contributes to the acidosis, by interfering with the efficiency of the body as a working machine, or by supplying it with too much fuel for the amount of work it has to do, is composed of a vast number of independent components. There is usually a combination of such factors as a diet which is faulty in respect of quantity as well as of quality, and an environment which fails to afford the required physiological stimuli for an active metabolism or one which actually damages the organism as a working machine by inflicting physical injuries upon it, as for instance in the way of causing some infective or inflammatory disturbance. In the accompanying diagram (fig. 5) I have attempted to provide a schematic picture of rickets which can be very easily visualized. At the bottom of the diagram are arranged in comprehensive groups some of the more commonly recognized aetiological factors that contribute their respective shares to the sum total of the acidosis which according to my view is the *causa causans* of the rickety condition.

Each individual item in the various groups of aetiological causes has its own individual symptom-complex, and each of them, or each combination of them, stamps the resulting clinical picture with its own individuality. Thus the clinical picture of rickets resulting from a deficiency disease such as scurvy will present a totally different appearance to one due to such contributory causes as carbohydrate excess or to a chronic catarrhal infection. Each clinical picture of rickets—in addition to the symptoms which are due to the acidosis and to the mineral depletion—will have the pathognomonic symptoms due to the particular combination of the contributory pathological conditions. By the use of a diagram of this kind, mentally visualized, it is possible to avoid confusing the symptoms which belong to the contributory aetiological factors, and those which belong to the acidosis itself, in other words to avoid a confusion between cause and effect. In this connexion it may well be asked, where does the picture of rickets begin, and where does the picture of the contributory conditions end? To this question I am not prepared to give a definite answer; but I cannot think that we are entitled to describe any case as one of rickets unless the contributory causes have definitely led to such a degree of chronic acidosis or mineral depletion that evidences of it are to be found in soft bones, muscular debility and nervous irritability.

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## Two Cases of Duodenal Obstruction in Infants.

By REGINALD C. JEWESBURY, M.D.

DUODENAL obstruction in children is a somewhat rare condition, and I therefore wish to place on record the two following cases which have recently come under my observation. Dr. Edmund Cautley has given a very complete account of this condition and has reviewed all the literature on it in an article entitled "Duodenal Stenosis" in the *British Journal of Children's Diseases*, 1919, xvi, p. 65. Cautley summarizes the symptoms as follows:—

"Vomiting, with the usual signs of obstruction, is the characteristic feature. It may even occur if no food is given by the mouth, the stomach becoming distended by normal secretion. Bilious vomiting occurs in about 90 per cent. of the cases, and if the obstruction is above the entrance of the common duct, is probably due to an aberrant branch opening into the dilated first part of the duodenum. Hæmatemesis is not uncommon. Naturally, inanition, wasting and constipation are marked features. If food is taken and life prolonged, as in some cases of stenosis, the stomach and first part of the duodenum become dilated and hypertrophied, and there is marked gastric peristalsis. The symptoms are practically the same as those of hypertrophic stenosis of the pylorus unless bilious vomiting is also present. . . . Many of these infants are premature."

### CASE I.—CONGENITAL STENOSIS OF DUODENUM.

Female child. History of vomiting in the mother during the last five months of pregnancy. The child born before this one died at nine days with a history of vomiting and constipation since birth.

Present illness: full term child, apparently normal; vomiting began third day; it vomited two or three times a day about one hour after feeds with the exception of the thirteenth, fourteenth, and fifteenth days. Vomiting projectile. The vomit was bile-stained. Bowels open about five times in all since birth, motions dark and small. Child never cried, slept all the time. Bottle-fed, diluted cow's milk. Nineteen days old when admitted to hospital, very wasted; weight, 4 lb. 9½ oz.; lies with legs drawn up. Since admission, vomited after every feed. Abdomen poorly covered, marked peristalsis from left to right. A rounded swelling extends from the left costal margin downwards below the umbilicus up to the right costal margin, at this point a small indefinite mass is felt. Peristalsis occurs every half to one minute.

X-ray report: "Dilatation of first and second part of duodenum and greatly distended stomach. Very little food had left in two hours. Probably duodenal obstruction" (fig. 1).

The child passed small infrequent motions and continued to vomit until death, which occurred on the twenty-ninth day.

*Abstract from Post-mortem Report.*— "Œsophagus normal. Stomach grossly hypertrophied, pylorus not thickened and pyloric lumen larger than normal. First and second parts of duodenum dilated and walls thickened owing to obstruction at third part.

Between the second and third parts of the duodenum is a much constricted portion about  $\frac{1}{4}$  in. long, which admits an ordinary probe. There is no evidence of compression from surrounding structures."

This appears to have been a case of true congenital stenosis of the duodenum.

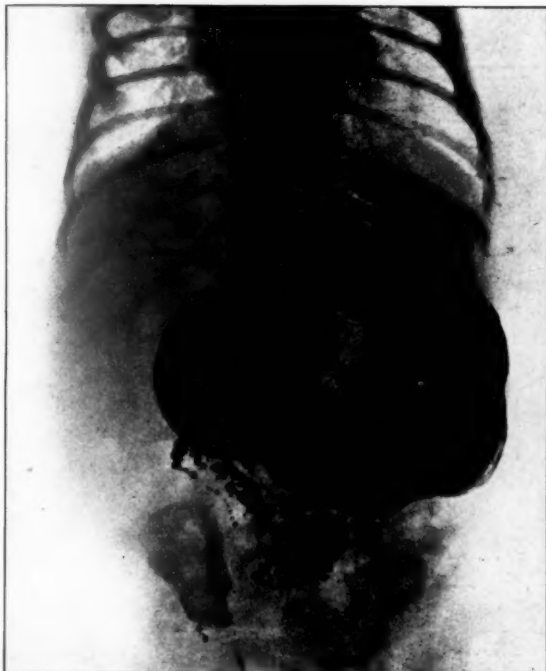


FIG. 1 (Case I).—Duodenal stenosis. X-ray two hours after bismuth meal showing dilatation of stomach, pylorus, and upper part of duodenum.

CASE II.—CONGENITAL OBSTRUCTION OF BOWEL AT DUODENO-JEJUNAL JUNCTION.

Male infant, aged 9 days. Family history: Parents healthy, one other child delicate. No miscarriage. Full-term child, breast-fed; bowels have been acting daily.

Present illness: Vomiting, projectile in character, had begun on the third day and had persisted since. The vomit was bile-stained. The child was rather wasted and slightly jaundiced, the stomach was dilated and showed peristalsis from left to right. The stools were intermediate in character between meconium and milk stools. After giving 2 oz. of breast milk by the bottle, the child was in evident discomfort and almost immediately vomited



## 12 Jewesbury: *Duodenal Obstruction in Infants*

with great force, the vomit was bile-stained and was more in amount than the small quantity taken. Vomiting occurred after every feed.

X-ray report—(bismuth meal): "Stomach retains most of meal for nineteen hours. Appearance suggests constriction of first and second parts of duodenum" (fig. 2).

Condition remained the same until death occurred on the thirteenth day.

*Post-mortem Report.*—The stomach and duodenum were enormously dilated and hypertrophied. Below the duodeno-jejunal junction the bowel was very

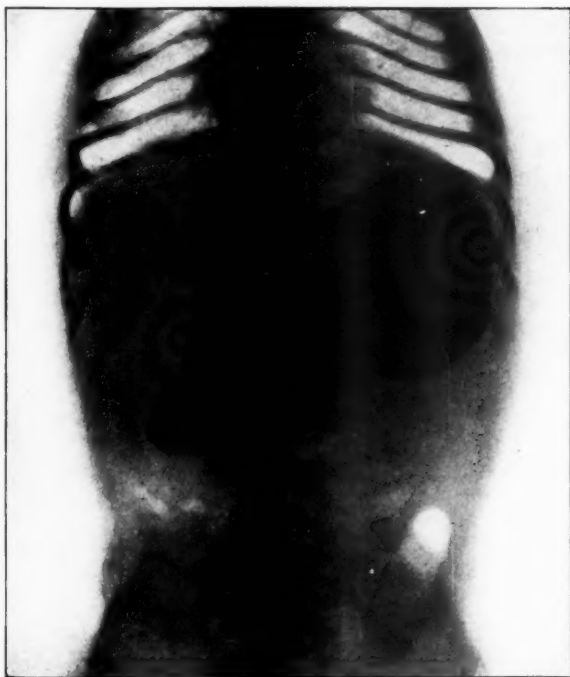


FIG. 2 (Case II).—Duodenal obstruction. X-ray two hours after bismuth meal showing dilatation of stomach and upper part of duodenum.

shrunk and small. The cæcum was situated abnormally high under the liver and the obstruction in the duodenum was caused by pressure from outside by surrounding structures and in particular by the right colic artery.

In this case, therefore, the obstruction was not due primarily to any abnormal narrowing of the duodenum itself, but to pressure on it from surrounding structures.

I am indebted to Sir Archibald Reid for the X-ray photographs.

## DISCUSSION.

Dr. PRITCHARD (President) said he had had one case of the kind in which he made a diagnosis of this condition, but as no post-mortem examination could be secured he was not able to verify it. The skiagrams now shown seemed extremely difficult to interpret, and he wondered whether other members thought so too. In the second case there was a peculiar shadow over the left side of the stomach, which seemed to him to merge into the shadow of the duodenum. He did not know what that shadow portended. A casual examination of one of the skiagrams led him to believe there might be some abnormality of the stomach, and several entries into the duodenum. Unless he had the support of some skilled interpreter, he would have been a good deal misled in that case. As the diagnosis was so important, he asked Dr. Jewesbury whether his diagnosis of the condition was made during life, or was it only revealed on post-mortem examination.

Mr. C. MAX PAGE remarked that Dr. Jewesbury had not said anything about the treatment of duodenal atresia, though he had pointed out the importance of diagnosing between duodenal atresia and hypertrophic stenosis. He would have thought that should be left to the surgeon. There might possibly exist some reason against operation, but it appeared to him that the only hope of the child surviving would be to subject it to operation, though admittedly the outlook would not be very bright, as gastro-enterostomy would be necessary, and the mortality from that operation was quite high. It would be interesting to hear Dr. Jewesbury's view as to the operative results in these cases; did he know what the statistics on the subject were?

Mr. R. A. RAMSAY said that one of the children described in Dr. Cautley's paper was operated upon by himself, but he did not get very far with it. The child was in a very weak state. The interest of the case lay in the fact that the child was nearly 12 months old, and it was difficult to understand how it had managed to reach that age, as the opening was so stenosed as to admit only a small-sized probe. There was no pylorus, and no constriction between stomach and duodenum. The duodenum was thicker than one's thumb, and at the stage he had reached he had to stop, as the patient was practically moribund.

Dr. JOHN THOMSON (Edinburgh) said that, ordinarily, in cases of pyloric stenosis the vomit did not contain bile, but he had seen three or four cases in which there was bile in the vomit, and in which the diagnosis was confirmed post-mortem. He remembered one case of congenital stenosis of the duodenum which was thought at first to be congenital hypertrophy of the pylorus, but differed in showing marked visible peristalsis on the fifth day. This usually occurred at a much later stage in the ordinary pyloric case. In that instance the child lived fourteen weeks. At the post-mortem examination the specimen appeared at first sight to be one of hour-glass stomach. Knowing the extreme rarity of that condition in infancy, when he was next at the Great Ormond Street Children's Hospital Museum, he asked to see any cases of hour-glass stomach which they had. He found that they had none, but there was one specimen exactly like his own which seemed at first sight to be that condition, but had turned out to be a case of the kind now described, the pylorus being enormously distended, so that it looked like the contracted part of an hour-glass stomach.

Dr. JEWESBURY (in reply) said there was considerable hesitation in making the diagnosis, but the X-ray reports and skiagrams were helpful, particularly in the first case. The X-ray interpretation of the second case was difficult. One helpful feature in arriving at a diagnosis was, that in these cases the vomit was bile-stained; it was so in both his cases. He was able to answer only the last part of Mr. Page's question. In the literature on the subject he did not find records of any operations having been performed for the condition. The only operation which, he thought, would be likely to benefit was gastro-jejunostomy. The first child was only a little over 4 lb. in

weight, and it was in a wretchedly weak condition, so that any radical operative procedure was obviously out of the question. Possibly if the case had been seen earlier and the diagnosis correctly made, operation might have afforded a chance of life. All seemed to depend on whether the child was strong enough to bear an operation. If he should meet with another case of the kind, he would be only too willing to call in his surgical colleague.

## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

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### Case of Cerebral Degeneration.

By HELEN INGLEBY, M.B.

I AM showing this case in the hope of obtaining opinions of members as to the category in which it should be placed. The patient was born in April, 1920. She had a fall on her head at birth, but recovered, and progressed normally until she was 20 months old, when she had an attack of broncho-pneumonia and whooping-cough. She is said to have recovered from this and to have started walking again. I do not think she ever walked very well; she pulled herself up by taking hold of things and walked a few steps at a time. She had another fall on her head about this date, but was, apparently, none the worse for it. Towards the end of last January, about a month after the attack of broncho-pneumonia, it was noticed that she began to fall about. This got worse until she could no longer stand, then she could not sit, her muscles wasted considerably, and swallowing was so difficult that ultimately she had to be fed with the bottle. Epileptiform fits started at Easter, and became more severe until she was having three or four, or more, in one day. Eventually she got into the condition you see now.

There are three other children in the family, all healthy; one was born subsequently to this child. There is no history of insanity, nor of tuberculosis, on either side. The father, who was in the R.A.M.C., wrote a very good account of the illness, and if there had been a history of syphilis probably he would have mentioned it.

On admission to the Victoria Hospital she was able to carry out all movements, but they were much weaker than those of a normal child of her age. There was general muscular wasting and laxity of ligaments. There were continuous aimless movements. She took no notice of what went on around her, and it was not easy to know whether she saw or heard. She appeared to have some perception of light, and the fundus was normal. She had great difficulty in swallowing. The abdominal reflexes were not obtained, and the plantars were extensor; deep reflexes were present. Sensations of pain were dimly appreciated. She had incontinence of urine and faeces. Lumbar puncture showed a great increase of pressure in the cerebro-spinal fluid. She was lumbar-punctured on several occasions at intervals of two or three days, and eventually the pressure became normal. The punctures were discontinued for a fortnight, and the pressure returned to the original height.

The fluid was examined by Dr. Stanley Wyard who reported a trace of albumin in the fluid, but no excess of cells or organisms. The Wassermann reaction was negative in blood and cerebro-spinal fluid. The electrical reactions were normal. She had a few fits after admission, for which she was given bromides, and the fits then ceased. She seems to have improved during her stay in hospital. Certainly her muscular power is now much better than it was, and she is able to swallow very much better. Probably the improvement is more apparent than real and has much to do with careful nursing.

As to the diagnosis, could there have been an encephalitis leaving this condition? Against this is the fact that the disease came on gradually; the only previous illness was broncho-pneumonia, from which she recovered. I do not see how we can fit this in with any form of encephalitis. The other possibility is, that it is a cerebral degeneration akin to amaurotic family idiocy, a late form of which has been recorded. It is generally familial, but sporadic cases are not unknown. Spasticity or pigmentation of the fundi are, however, characteristic of these cases and neither of these is present here. I have not, so far, come across a case in which the symptoms started at the age they did in this child.

Dr. BELLINGHAM SMITH thought this was a case of progressive cerebral degeneration rather than of either encephalitis lethargica, or encephalitis following an acute infective disease. The history made it clear that the child recovered from whooping-cough, and walked about afterwards, and that the onset of the weakness and general mental degeneration had been slowly progressive. The rolling of the head and eyes, suggestive of blindness, and the slow mental and physical deterioration, led him to think that it was one of those cases the pathology of which was still unsolved, allied to amaurotic family idiocy but differing in not having the typical fundal picture, and not necessarily occurring in Jewish children.

### Double Tumour, ? Lipoma, in the Perineal Region of an Infant.

By BERNARD MYERS, C.M.G., M.D.

P. S., AGED 3½ months, was brought to the Royal Waterloo Hospital when 1 month old, on account of two small swellings in the perineum. The larger was about the size of a pigeon's egg, and the other a little smaller. They were situated about ¾ in. in front of the anus and to the left of the middle line. The tumours were sessile, of smooth surface, and covered with skin. The consistency rather suggested that they were of a fatty nature. The mother said that the tumours were present at birth. Since first seen the growths have both distinctly increased in size to about that of a tangerine orange, and they now both spring from a common pedicle. The opinion of the Section is asked (1) as to their probable nature; (2) whether excision should be performed at once? The child is otherwise normal and healthy.

#### DISCUSSION.

Mr. MAX PAGE said he considered that the tumours were soft fibromata; there were definite narrow pedicles, and he would cut them off. He did not regard them as dermoids, or as any form of developmental abnormality.

Dr. ERIC PRITCHARD (President) said that most of those who examined the case thought the condition was lipoma. It was agreed that removal was the best treatment.

### **Case of Persistent Jaundice in an Infant; Atresia of the Common Bile-duct and Biliary Cirrhosis.**

By BERNARD MYERS, C.M.G., M.D.

G. W. P., AN infant, aged 7 weeks, was brought to the Royal Waterloo Hospital by Dr. Outram, of Edmonton, on July 7, 1922, and admitted to the ward. Persistent jaundice had begun two days after birth, becoming more insistent during the second week, and had since continued. The motions were white, and the urine distinctly bile-coloured. On the napkins obvious bile-staining was apparent. The infant was fed on breast milk and glaxo, which it took well. It had otherwise been enjoying good health.

Family history: Icterus neonatorum had been present in the mother's three other children, disappearing in four or five days, but there was no case in her family similar to this one. She had not had any miscarriages.

No malformations were demonstrable when the child was examined, and the nutrition seemed to be quite satisfactory. The infant weighed 8 lb. 4 oz. The skin was distinctly jaundiced and also the conjunctivæ. Enlargement of the liver to the extent of 2 in. below the costal margin was observed, but it was not possible to feel the spleen. If gentle pressure was made over the right side of the liver the child seemed to be in pain, but this was not observed after the first week in hospital. The heart and lungs were apparently normal.

There had been continuous vomiting since admission; but food had been satisfactorily taken. There was a gain of weight until July 14, but a loss of 8 oz. since admission was discovered on July 21, and the child, still jaundiced, did not look quite so well. The condition became generally worse with a loss of weight, and the liver could no longer be palpated below the costal margin. Death took place on August 23, when the child was aged 14 weeks. Wassermann reaction negative; no signs of syphilis. No hæmorrhage had been observed from the skin or mucous membranes during the whole time the child was in hospital, and there was no history of such hæmorrhage before admission.

The jaundice became less marked during the last two weeks of life, though always distinctly evident, especially so on the conjunctivæ. The bowels acted two or three times daily, occasionally there were four or five motions in twenty-four hours. The temperature was either normal or slightly subnormal and the pulse varied from 100 to 145.

Mr. J. A. Cairns Forsyth states: "Since the outlook without operation is hopeless, exploratory laparotomy is justified on the off-chance that the obstruction may be low down in the common bile-duct and anastomosis feasible. But these patients make bad subjects for operation on account of their tender age, and the risk of hæmorrhage. Only in about 16 per cent. of these cases are the gall-bladder, cystic and hepatic ducts pervious to the flow of bile."

Dr. T. G. Shore reported on the liver as follows: "The liver is firm and of a dark green colour. The anterior margin is sharp. The surface is smooth,

but somewhat tuberculated, raised areas being separated from one another by linear depressions, which have a branching arrangement, and are doubtless due to the close relation some of the portal spaces have to the surface of the liver. After a short preservation in Kaiserling's solution the weight of the liver is 152 gr., which is probably not materially different from its weight in the fresh state. Anatomically the lobes of the liver are natural. The gall-bladder is small, cylindrical, and thin walled, and contained a small quantity of dark green bile. The hepatic ducts are not distended. The openings of the gall-bladder into the cystic duct, and of this into the common bile-duct are demonstrable, but the lumen of the common duct gradually tapers until it can be traced no longer, and the duct is lost in a mass of toughish fibrous tissue half an inch or more from what should be its termination. The portal vein and hepatic artery are easily dissected, and are normal. The small portion of the pancreas left attached to the specimen contains the duct of Wirsung, which can be traced to a papilla on the mucous membrane of the duodenum situated about  $1\frac{1}{2}$  in. from the pyloric sphincter. This doubtless represents the ampulla of Vater. No other duct joins that from the pancreas at this point. The pancreas presents a normal appearance, except some staining with bile pigment, a condition shared by the duodenum.

"Microscopic examination of the liver shows distension of the biliary radicles within the lobules. These small ducts contain plugs of dark green material, and in places the adjacent liver tissue is stained in a similar manner. Some of the liver cells are small, and their nuclear staining is poor, but on the whole the cells are well preserved. The portal spaces are prominent; the bile ductules are distended and contain the same dark green material as the bile capillaries; the arteries are thickened by an increase of the tissue of their middle coats, and the branches of the portal vein are similarly affected. The connective tissue of the portal spaces is increased in amount, and is markedly fibrous. Such thickened portal spaces can be seen lying at the bottom of fissures from the surface, as described above. Fibrosis is limited to the neighbourhood of the portal spaces."

The cirrhosis of the liver most likely followed the atresia of the common bile-duct, and not vice versa.

Dr. WYARD said that he had published in the *Lancet* in 1914<sup>1</sup> an almost identical case, except that in his case the condition was even more marked, as there was no common bile-duct at all, and no cystic or hepatic ducts could be found. The gall-bladder was a mere fibrous cord. In that case, operation was out of the question, as there was nothing which could be brought down along which the bile could come. In the liver itself there was definite biliary cirrhosis, with attempts at the formation of new bile-ducts, and the fibrosis extended into the lobules, which were practically destroyed. The cellular degeneration was much more marked than in this case. Those bile-ducts which could be found were full of a substance which was taken to be bile. The child lived fourteen months.

<sup>1</sup> *Lancet*, 1914, ii, p. 495.



## Case for Diagnosis.

By BERNARD MYERS, C.M.G., M.D.

P. L., AGED 3½ months, was brought to the Royal Waterloo Hospital when aged 16 days. Upon examination both elbow-joints were found to be extended. forearms pronated, wrists and fingers flexed, the thumbs being inserted under the fingers towards the bases of the little fingers. The limbs were rigidly held in this position slightly in front of the body. Attempts made to place the thumb in a normal position evidently caused pain and the child cried at once. Still it was possible gradually to overcome the spasm of the fingers and wrists. The knee-joints were extended and the toes pointed, the heels being drawn up. A decided degree of flexion was present at the hip-joints, the lower extremities being thus placed toward the front of the body. It was very difficult to overcome the spasms of the ankle-joints and toes. The reflexes could not be properly tried. There seemed to be some abnormality of the left elbow and both knee-joints. Dr. Tindal Atkinson reported that the skiagram showed no abnormality of any of the bones or joints. Wassermann reaction negative. Mentally the child is particularly bright and alert, and the cranial nerves appear to be acting normally. The muscles around the shoulder and pelvic girdles are very small and apparently weak. All the muscles of the upper arm, and thigh more particularly, and those of the forearm and leg to a lesser degree, show a certain amount of atrophy. The muscles of the back seem to be less affected. The abdomen is distended. The muscles appear to act normally to galvanism and faradism. The child lies always to one side or the other. The fontanelle was 1 f.b. Dr. Bickerton reported no changes in the discs or fundi. Cerebro-spinal fluid normal. The child has not gained in weight since admission. The appetite is satisfactory and the bowels act daily. The temperature has been normal throughout, with the exception of the first few days, when there was a slight rise.

A possible diagnosis was that of decerebrate rigidity, but Dr. Kinnier Wilson, who kindly examined the case, is of opinion that there are certain difficulties in the way of that view. The opinion of the Section is asked as to the diagnosis and any treatment that should be suggested with regard to the deformities of the elbow and knees, and to rectify the position of the feet and hands.

Dr. G. A. SUTHERLAND pointed out that there was a peculiar absence of affection of the trunk and neck muscles. Decerebrate rigidity was usually more general than the condition in this child. Some years ago he had a case of decerebrate rigidity, to which he gave the name cerebral aplasia. Some years afterwards he had a second case, and the symptoms were so exactly reproduced in this second case that he felt sure of the diagnosis. All the muscles were more or less stiffened in those two cases, and though there was also marked opisthotonos, on looking at the face there seemed to be nothing wrong. The underlying condition was one in which the cerebral hemispheres had not developed at all, and at the autopsy, free fluid occupied the space which ought to have been taken by the cerebral hemispheres. It was possible that the lack of development in the cerebral hemispheres was not complete in every case, a fact which could not be expected to be revealed during life. With regard to treatment, the lesion was a cerebral and congenital one, therefore treatment was not hopeful, and he would not recommend even an orthopaedic appliance; in fact he would postpone doing anything *sine die*.

**Case for Diagnosis; ? Renal Dwarfism.**

By G. M. KENDALL, M.B.

THE patient is a girl aged 4 years and 6 months who had been apparently healthy up to the age of 2 years and 6 months. She then had an acute illness and was admitted to the Victoria Hospital for Children. Her condition was as follows: There were general œdema and ascites which appeared and reappeared several times during her stay of four and a half months. In addition there were diarrhoea and vomiting at first: and this also recurred at intervals. Urine, on repeated examination, contained no albumin. Wassermann reaction of blood positive; Wassermann reaction of mother positive; of father negative. Patient was treated with mercury, and a course of N.A.B. intravenous injections was given.

Since discharge she has had, at intervals of a few months, attacks of illness. She becomes swollen all over and has sickness and diarrhoea. She was again admitted, during one of these attacks, on October 25, 1922. For six months past knock-knee has been noticed. During about a year she has drunk rather large quantities of fluids, and at times, but not constantly, has had polyuria.

No history of serious illness or deformity amongst her relatives.

On readmission she was well nourished and appeared intelligent for her age. Height, 36 in.; weight, 32½ lb. There was considerable œdema of the legs and thighs, and some ascites. She had moderate diarrhoea and vomited occasionally. Both the œdema and the gastro-intestinal symptoms have varied while she has been in hospital. Once for a day or two the whole body and face were very much swollen, but at times there is only a slight œdema of the shins and feet. Carpal spasm has been noted, and Chvostek's sign is present.

The urine at first showed no albumin. Recently on several occasions there has been a faint trace of albumin; no casts have been seen; the quantity cannot be determined, but does not seem excessive. The cardiovascular system is apparently normal; blood-pressure 80/55. There are no abnormalities in the fundus of either eye.

Results of renal efficiency tests: Blood urea (on full protein diet), 11·5 mgr. Urea concentration test (10 grm. given): First hour, 1·2 per cent.; second hour, 1·8 per cent.; third hour, 1·6 per cent. After 2 grm. of potassium iodide by mouth, 48·8 per cent. was excreted in the first twelve hours and 2·6 per cent. in the second.

The skeletal system shows no gross deformity except well marked genu valgum. The lower epiphyses of the radius and ulna appear enlarged. The X-ray plates show that the ends of the diaphyses of the tibiae and femora are translucent and striated longitudinally; the epiphyseal line is straight; there is no cupping of the diaphyses as in rickets; the ends of the diaphyses of the long bones of the upper limbs are also translucent but more irregular: there is not the same longitudinal striation and the epiphyseal line is not straight. There is periosteal thickening of the shafts.

The Wassermann reaction is now negative.

The first point to be decided is whether this case belongs to the group described as renal dwarfism. The evidence of renal disease is not very conclusive, but the X-ray appearances seem similar to those published by Barber in the *Quarterly Journal of Medicine* for April, 1921.

The next point is whether the congenital syphilis which is probably present here has any ætiological significance. In the published series only one case had a positive Wassermann reaction. It seems that we are dealing with two diseases in the same patient.

It will be noted that this patient is only an inch or two below the average height of her age. In most of the other cases the dwarfing was very marked, but the deformity had appeared later in life and the patients were older when first seen.

In conclusion I have to thank Sir Humphry Rolleston for allowing me to show this case.

#### DISCUSSION.

Dr. PATERSON said that although the skiagrams were like those in the cases published by Dr. Hugh Barber<sup>1</sup> and himself,<sup>2</sup> he regarded the present case as primarily intestinal. Possibly it might be Hirschsprung's disease. He believed it might fit in with the picture of tuberculous ulceration of the bowel, a condition in which he had seen œdema, attacks of tetany, and bursts of diarrhœa. It was, he thought, primarily a bowel, not a kidney case.

Dr. G. A. SUTHERLAND agreed with Dr. Paterson that this was primarily an intestinal case, and dwarfism could be left out of account. He did not however agree that there was tuberculous ulceration; if there had been, he did not think the child would have been looking so well as it did now. He did not know whether this was not merely a condition of gastro-intestinal disturbance with great dilatation, such as was sometimes met with in rickets, and which would explain all the symptoms. There was, he thought, considerable disturbance in the colon, and treatment should be directed to that condition.

Dr. E. BELLINGHAM SMITH said he also agreed that this was a case of gastro-intestinal disturbance, but that it did not look to him like a tuberculous case. He suggested that the gastro-intestinal trouble was in some way dependent on the syphilis: there was some diffuse syphilitic infection of the intestinal secretory glands, and of the bowel itself. He would have liked to hear whether there was anything in the stools suggesting pancreatic or any other glandular insufficiency. Tetany was commonly associated with chronic gastro-intestinal disturbance and a moderate degree of œdema of arms and legs was not unusual where the tetany was pronounced and of long duration.

Dr. F. PARKES WEBER said the case reminded him of the case of a girl, aged 8 years, shown some time ago by Dr. F. Langmead, in which there was tetany connected with dilatation of the colon.<sup>3</sup>

Dr. KENDALL (in reply) said that the stools had been examined bacteriologically, but not chemically; no pathogenic organisms had been discovered in them. The stools were not large.

<sup>1</sup> H. Barber, "Renal Dwarfism," *Quart. Journ. Med.*, 1922, xiv, p. 205.

<sup>2</sup> D. Paterson, "Cases of Renal Dwarfism," *Proceedings*, 1921-22, xv (Sect. Study Dis. Child.), p. 4.

<sup>3</sup> F. Langmead, "Facial Irritability of Fifteen Months' Duration succeeding Tetany, in Association with Dilatation of the Large Intestine," *Proceedings*, 1909, ii (Sect. Study Dis. Child.), p. 218.

## Two Cases of Celiac Infantilism in the Convalescent (Non-diarrhœic) Stage.

By REGINALD MILLER, M.D.

THESE are exhibited to demonstrate (1) that in the symptomless convalescent stage of celiac disease analysis of the stools will still show an excessive fat-wastage; and (2) that if the excess of faecal fat is chiefly in the form of soaps, the stool may be dark, even constipated, and far from fatty in appearance.

*Case I.*—D. F., born October, 1911. Severe celiac symptoms from 1913 to 1920, with pale diarrhœa, enlargement of the abdomen, severe anorexia and periods of refusal of food and marked stunting of growth. Developed late rickets at the age of 7 years. (Published up to January, 1920: *Lancet*, 1920, ii, p. 894.) At that date was put on a constant diet and analyses made of faecal fat before and after administration of bile-salts and alkalies (analyses A and B). Since this date improvement has been uninterrupted, and since September, 1920, the stools have been persistently dark and formed, sometimes constipated. The appetite is excellent and child has grown 4 in. (present height 3 ft. 5½ in.). She has gained 14 lb. The stools still show an excess of fat, now chiefly in the form of soaps (*vide table*).

*Case II.*—G. W., born October, 1909. Celiac symptoms from 1911 until January, 1920, much stunting of growth, enlargement of abdomen and severe pale diarrhœa. First seen in April, 1920, after three months absence of diarrhœa; growth had then restarted. Since then she has continued to grow well (5 in. in two and half years, now 4 ft. 2 in.), and has had no diarrhœa. She is free from symptoms except that the stools are sometimes pale. She has been on ordinary diet all the time but this is sometimes too rich in fats for her. Even when constipated the stools still contain an excess of fat.

TABLE OF FÆCAL ANALYSES.

	Date	Percentage of fat in faeces	Per 100 grm. faecal fat			Appearance
			Neutral fat	Fatty acid	Soap	
<i>Case I.</i>	January, 1920 (A)	52.4	15.3	75.4	9.3	Pale
"	January, 1920 (B)	33.7	7.7	49.3	43.0	Coloured
"	March, 1921	26.8	8.9	37.4	53.7	Dark
"	June, 1922	33.2	7.2	40.9	51.9	Dark
<i>Case II.</i>	March, 1921	39.6	7.0	28.3	64.7	Dark, hard
"	October, 1922	56.4	5.7	50.4	43.9	Coloured, greasy

I wish to call attention to two points. One is the condition of the celiac child in the convalescent stage. We often talk about celiac disease "clearing up," but it seems that the mal-absorption of fat, the essence of this condition, still remains, even when the symptoms cease. I show these cases to emphasize that point. You may see a case of celiac disease pass from the acute stage, with pale diarrhœa, large abdomen, loss of appetite, and cessation

of growth, to the convalescent stage, when there are dark formed stools, and resumed growth; yet analysis will show there is still great fat-wastage going on. That is well shown in both these cases (*see* Table). Therefore, if we do not know when coeliac disease ends, it is possible that we do not know when it begins, and some of our dates, putting the origin of coeliac disease down to an acute attack at the age of 18 months, may be wrong; there may have been defective fat absorption long before that.

The second point concerns the explanation of this; how is it that a patient can be free from symptoms and passing what appear to be normal stools yet containing excessive fat? The answer is, that in the non-diarrhoeic stage the fat is present chiefly in the form of soaps. That is a point which was new to me until a year or two ago.

Thus we see that there are three types of fatty stool. First, the type of pancreatic insufficiency, with a large excess of fat in the stools, chiefly in the form of neutral fat. This condition never arises in coeliac disease. Secondly, that of excess of fat chiefly in the form of fatty acids giving the classical coeliac stool, large, pale, unformed and very offensive. Thirdly, there is the soapy stool, looking normal upon casual inspection; it is not offensive, and not frequent, yet it may be found to contain anything up to 50 per cent. of fat, chiefly in the form of soaps. This is the stool that is found in the non-diarrhoeic stages and cases of coeliac disease. The only obvious alteration in it from the normal is its increased bulk.

With regard to treatment: as the patients who pass their fat chiefly in the form of soaps do well and remain free from symptoms, we should try to do two things: (1) To convert fats into soap; (2) to increase the fat absorption. Since I have worked on these lines my results have been conspicuously better than they were before, and I do not know any disease which responds more satisfactorily to treatment than this when those two points are kept in view.

#### DISCUSSION.

Dr. F. PARKES WEBER asked under what name these cases were known on the Continent. Also whether, in fatal cases, the patients died because the treatment was wrong, or in spite of good treatment.

Dr. PATERSON asked for more details of the treatment adopted by Dr. Miller in these cases; points, for example, about diet and the medicinal treatment.

Dr. MILLER (in reply) said very little work on this subject had been done on the Continent. In this country, at one time, the condition was regarded as pancreatic infantilism, and in America and on the Continent it was usually called intestinal infantilism of Herter. The treatment he adopted was as follows: he analysed the stools on ordinary diet and ascertained the percentage of fat. Then he gave a diet as free from fat as possible, e.g., milk skimmed by scalding for twenty minutes, letting it stand for four hours, and removing the cream-clots. He replaced butter by syrup, anchovy paste, &c., giving lean meat and fish. The patient was kept on that diet, until a certain improvement was reached—i.e., until the colon was no longer flatulently distended; the toxic, grey complexion was changed to a more ordinary pallor, and the appetite for protein and carbohydrates returned. If the patients were sufficiently starved of fat, they could be brought to eat their other food properly. At the end of that stage he again analysed the stool, and the lowest minimum of fat passed in the stools was ascertained. Then he gradually re-introduced fat into the diet, commencing with very small "scrapes" of butter, and going on to puddings made with unskinned

milk, and if the patients did well, with milk partly skimmed. He aimed at keeping the percentage of fat in the stools between 25 and 35 per cent.; at that stage the patients would begin to put on weight again. Then he gave them bile salts, washing these down with an alkaline drink—rhubarb and soda, or magnesia or calcium. He gave alkalis from the beginning and throughout, in the hope of aiding the saponification of the fats. If, after giving the bile salts, the fat in the stools dropped, a little more fat was allowed in the diet. The patients should be kept resting during the earliest stages of the treatment, lest they lost weight too rapidly.

(Dr. Miller demonstrated the weight-chart of a case of coeliac disease in the diarrhoeic stage coming under treatment for the first time at the age of 2½ years. There was a gain of weight of nearly 4 lb. in four weeks.)

### Hereditary Tylosis.<sup>1</sup>

By J. D. ROLLESTON, M.D.

THE patient, a girl, aged 2 years, presents a symmetrical thickening of the epidermis of the palms and soles. The thickening ends abruptly at the border of the palmar and plantar surfaces, and is separated from the normal skin by a narrow pinkish halo. The condition was first noticed by the mother a few months after birth, and has persisted ever since. Exfoliation of the palms and of the soles occasionally takes place. The nails are not affected, and there are no skin lesions elsewhere. Apart from a slight degree of epicanthus the child does not present any other abnormality.

The only other child in the family, a girl, has normal palms and soles. The father, and one of his sisters, both of whom I have seen, present tylosis of the palms and soles in a very marked degree. They state that another brother has a similar condition, and that this child is of the fifth generation affected, but are unable to give any details as to previous generations. The child's mother is not affected. This condition is well-known to dermatologists, but there is little to be found about it in pædiatric literature.

The child was in hospital two months on account of diphtheria and whooping-cough, and the condition of the skin puzzled my colleagues and myself. It was only two days before the child went out that I read an article by Dr. Aronstam,<sup>2</sup> of Detroit, Michigan, on a condition he described as *keratoderma palmaris and plantaris hereditaria*, when I became convinced that this was a case of that sort. I call it by the convenient name of tylosis; sometimes it is known as congenital ichthyosis.

Treatment for the condition appears to be only palliative; a number of applications have been tried in various cases, such as salicylic acid, and soaking the hands in water, and even the application of X-rays. But the condition relapses very much. Apart from some blunting of the sensibility, not much inconvenience is caused, unless it is in those who engage in severe manual labour, as, for example, the patient's father, whose hands have become very sore.

<sup>1</sup> The case will be published in full in the *British Journal of Children's Diseases*, January-March, 1923.

<sup>2</sup> *Urological and Cutaneous Rev.*, 1922, xxvi, p. 550.

DISCUSSION.

Dr. F. PARKES WEBER said he supposed no one would object to including these cases as examples of a kind of *nævus*. *Nævus* of various kinds might sometimes be distributed unilaterally, or in a segmental way; but in these cases it was distributed in an acroteric way, i.e., it was a kind of *acro-nævus*. He wondered whether Dr. Rolleston had met with cases of congenital keratosis in which the superficial blood-vessels were also congenitally affected, cases which had been described as congenital erythro-keratosis or erythro-keratoderma.

Dr. ROLLESTON replied that, so far as he could gather from a study of the literature, the pathogenesis of this condition was obscure. The *nævus* theory had been raised. He had never seen a case of erythro-keratosis.

**Case of Rickets treated by Light Therapy.**

By R. C. JEWESBURY, M.D.

THE patient is a negro child, now  $2\frac{1}{2}$  years of age; the father is a West African, the mother is a white woman. The child came under observation four months ago, presenting then well-marked signs of active rickets. It had been breast-fed nine months, but had been living under very unsatisfactory conditions, and the diet was not suitable. As I thought it would be a good



FIG. 1.—X-ray taken before treatment showing well marked rachitic active changes.

case upon which to test the value of artificial light as a curative agent, I took the child into hospital, and put it on a diet containing a minimum quantity of fat; the milk was limited to half a pint. It had margarine instead of butter, and no other fat-containing food. The child was kept indoors in the ward throughout this treatment. It was treated daily by Dr. Murray Levick, the whole body being exposed to the radiations from a carbon arc lamp. The



X-ray photographs, taken at fortnightly intervals, show the progress very well; there is an interval of six weeks between the first photograph reproduced and the last. In the last, the rickets is seen to be almost healed, and the case seems to prove the very great value of artificial light in this condition.



FIG. 2.—X-ray from same case taken after six weeks' treatment by carbon arc light, showing very marked improvement.

### **Pneumococcal Septicæmia and Enlargement of Liver and Spleen.**

By R. C. JEWESBURY, M.D.

THIS case is interesting from the point of view of diagnosis. The child is  $3\frac{1}{2}$  years of age, and she came to hospital with a history of only two days of abdominal pain. The abdomen was tender and somewhat distended. The liver and spleen were very considerably enlarged, the spleen, in proportion, being larger than the liver. A moderate degree of ascites was present. When admitted, her temperature was  $104^{\circ}$  F., and the chart since shows an irregular pyrexia. As she looked so anæmic, a blood examination was made and showed a reduction of red cells to 3 million, the hæmoglobin was 55 per cent., the leucocytes were over 12,000. The differential count shows nothing in particular. A blood culture showed a pure growth of pneumococcus; therefore the case is one of pneumococcal septicæmia. Wassermann reaction: negative.

From the point of view of diagnosis, it is difficult to see what relationship the blood condition bears to the abdominal state. The liver feels like a cirrhotic liver. Are there here two distinct conditions: septicæmia of recent origin, and a condition of some chronic enlargement of liver and spleen? If so, what is the nature of that enlargement? I suppose it is possible that it is cirrhosis of the liver combined with cirrhosis of the spleen, causing enlargement of both; that hypothesis might account for the ascites. The heart is of normal size, but the first sound is not quite pure in character, though there is no definite murmur.

## Three Cases of Inborn Errors of Metabolism.

By DONALD PATERSON, M.B.

## (I) CONGENITAL STEATORRŒA.

PATIENT, a male, aged 3 years, was brought to the out-patient department of the Hospital for Sick Children, Great Ormond Street, London, with the complaint that he was passing fat in his stools. Patient is one of nine children, all well: both parents appear to be healthy.

This child has had no previous illnesses, but it was noticed that when he was weaned from the breast and put on "glaxo," the motions contained fat which "set" after being passed. The motions have been brown in colour ever since, whatever the kind of food taken by the child. Occasionally he passes several stools during the day, but usually only one, and it is never formed.

On examination: Size and weight are normal and he has a good colour; no abnormalities were found in the various systems.

The motion seen looked like thick yellow vaseline and had a strong smell like that of cheese or butter. It was quite oily and left a greasy mark on paper. The analysis of two stools showed that more than 99 per cent. was composed of fat, of which only 8 per cent. was split.

The Wassermann reaction is negative.

## (II) TWO CASES OF CONGENITAL CYSTINURIA.

Two children in the same family, aged 4 years and 2 years respectively, continually pass cystin in the urine. The elder had a calculus removed from the bladder when he was 2 years old; the younger has had a bladder calculus removed quite recently. Both calculi were composed of cystin.

An attempt is being made to control the formation of cystin by giving a low protein diet, and to prevent the cystin from being precipitated by giving sodium bicarbonate to keep the urine alkaline.

## DISCUSSION.

Dr. REGINALD MILLER said that only two cases of congenital steatorrhœa had been recorded: one by Sir Archibald Garrod and Dr. Hurlley,<sup>1</sup> and the other by the speaker and Dr. Perkins.<sup>2</sup> A third had been mentioned by Dr. Mackenzie Wallis.<sup>3</sup> Sir Archibald Garrod called his case congenital *family* steatorrhœa, and the parents of that child were cousins. In his own case, a sporadic one, the parents were not related before marriage. The condition seemed to be due to a congenital absence of lipase; it did not interfere with the child's growth. These patients passed the typical pancreatic stools, in which, on a rich diet, the fat was separated from the faeces—the condition known as "butter stool." He thought the specimen referred to by Dr. Paterson, with 99 per cent. of fat, must have been the separated fat, not the faeces. The passage of fat could be diminished by dieting, and, as in cœliac cases, one must

<sup>1</sup> *Quart. Journ. Med.*, 1912-13, vi, p. 242.

<sup>2</sup> *Ibid.*, 1920-21, xiv, p. 1.

<sup>3</sup> *Loc. cit.*, p. 86 (Case 8).

work out the diet which contained the optimum percentage of fat: then the patient no longer suffered from the incontinence of oily faeces. The other point about treatment concerned pancreatic preparations; as far as he knew there was no pancreas preparation on the market which contained lipase, and that was a cause of disappointment.

Dr. F. PARKES WEBER remarked that Sir Archibald Garrod's case (which had previously been under Dr. K. Fürth's care at the German Hospital in London) seemed to have been absolutely the first recorded case of congenital steatorrhœa.

## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

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### EXHIBITION OF CASES SHOWING THE LATE RESULTS OF ENCEPHALITIS LETHARGICA, FOLLOWED BY DISCUSSION.

#### (A) Cases.

##### (I) R. C. JEWESBURY, M.D.

I HAVE brought two cases for examination. One is that of a boy now aged just over 4 years. He was a perfectly normal child up to the end of his first year. He then had some indefinite febrile illness, and I did not see him at the beginning of it; I was told that during that illness he had progressive weakness of his limbs, and became unable to crawl, although he had crawled previously. He was also unable to sit up, or even to hold up his head. There was no history of fits. He was admitted to St. Thomas's Hospital, at the age of 15 months. He then showed marked hypotonia, the tendon reflexes were absent, there was a slight internal strabismus, and he was unable to sit up unaided, or to hold up his head. Apart from that, there was nothing abnormal.

As to his progress: He developed at times a tetany position of the hands. The cerebro-spinal fluid appeared to be quite normal, and his Wassermann reaction was negative. After being in hospital a month he was discharged. That was three years ago.

His present condition is as follows: There is a remarkable spasticity of all his limbs, he is still unable to sit up, and has no idea of standing; his mother now finds it difficult to get him about, as he is a heavy, well-developed child. All his reflexes are exaggerated, and he has an extensor plantar response on both sides. The most striking feature about this case is that although there is this remarkably spastic condition, the mentality is extraordinarily clear.

The other case I have shown is that of a girl, aged 10 years. In 1918 she had what was thought to be an attack of meningitis. I did not see her at that time, but after that attack she lost power in the limbs, and was unable to speak for five months. She gradually recovered from both those disabilities, but was left with a marked inco-ordination, and ataxy; the muscular tone was increased. When seen at that time there was but little to be made out in the nervous system. The plantar reflexes were flexor, and the tendon-jerks were rather brisk, but there was nothing abnormal about the cranial nerves, and the mental condition was good. The most prominent features were ataxy, and inco-ordination. That was four years ago.

She still shows distinct ataxy, she can walk alone, but is very unsteady, and sometimes falls down. She has an intention-tremor of the hands, her speech is slow, and although at the time of her illness she was mentally very clear, she is now distinctly backward. She attends a special school, and the authorities there regard her as a very backward child.

In a third case, the patient, a boy, now aged 8 years, had an attack of encephalitis two years ago, for which he came into hospital. I saw him again yesterday and except for a slight tremor of the hands he now appears to be quite normal.

(II) H. C. CAMERON, M.D.

*Case I: Encephalitis Lethargica, 1917; Attacks of Hyperpnœa, Mild Parkinsonian Type.*—A. B., aged 6 years. Admitted to Evelina Hospital, 1917, for an acute illness with pyrexia and unconsciousness. Discharged to infirmary three weeks later with measles. Discharged from infirmary at the end of a month, unable to speak, became babyish in her habits and for long took no notice of her mother. At times very restless and excited, and complained of severe pains in the head. Never had any difficulty in swallowing. Frequent attacks of hyperpnœa, breathing very noisy, and face flushed. Has transient attacks of unconsciousness, falls down and bruises herself. Was sent away from school for this. She is quick, plays with toys and dolls, but is destructive and has cut off her sister's hair. Affectionate at times. Frequent nodding movements of head to and fro. Face a little stiff and blank at times. Outstretched arms and hands tend to be over-extended and show slight tremor. Tendon reflexes all increased. Equilibrium not very good, easily pushed over. Speech still difficult and she is often unable to get the words out. Very shy. On the whole she is improving.

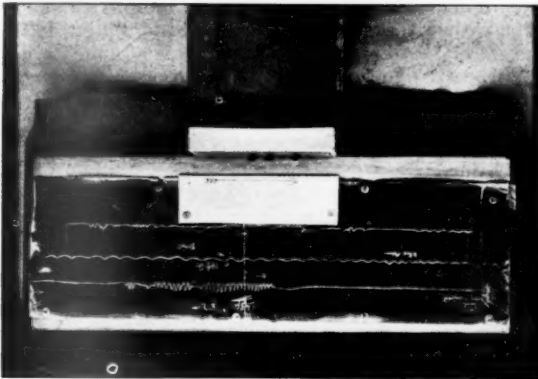
*Case II: Encephalitis Lethargica, 1918; Attacks of Hyperpnœa: Slight Mental and Physical Deterioration.*—F. B., aged 6½ years. Both patient and his younger brother had "sleeping sickness" in 1918. Stupor for ten days, and unable to walk for two months afterwards. Since then has had frequent attacks of noisy hyperpnœa, lasting for two or three hours at night. The noise can be heard in the next room. Does not speak plainly, not so well as his younger brother aged 5 years. Is quite good, not spiteful. Walks well but gets tired easily, complains that his knees and his legs give way. Equilibrium not very good. Behaves well but is not so sharp as his brother. Physical examination reveals nothing abnormal. Brother appears to be quite unaffected.

*Case III: Encephalitis Lethargica, April, 1918; Spastic Paralysis, Gross Mental Deterioration.*—A. L., aged 5 years. Only child. Normal labour. At the age of 3½ months admitted to King's College Hospital with a pyrexial attack and coma. The coma lasted three months. Diagnosed ? botulism, ? encephalitis. Remained very ill and stuporose for about a year. Sat up at the age of 2½ years and has only walked without support for the last six months. Increased tendon reflexes in arms and legs but much more so on left side. Left side of face also involved. Paralysis of palate and strabismus. Dribbles a great deal and regurgitates fluids. Sleeps well. Very emotional, no reserve, will bite at strangers. Some evidence of self-protection. Takes no notice of toys. Does not feed himself. Can just say "Mamma." Mother thinks he understands what is said to him, e.g., simple commands. Has not at any time had any peculiarity of respiration. Progress doubtful.

*Case IV: Tracing showing the alternation of Hyperpnœa with long Apnœic Pauses in a child aged 16 months.*—W. R., aged 16 months. Full time; normal labour. No previous illnesses. If this child had had encephalitis lethargica it had been so mild that it had passed unnoticed. For the last

seven months he had been subject to phases of restless excitement. Somnolence was not a prominent feature. At times there were attacks of hyperpnœa, with loud noisy breathing, flushed face and anxious expression. As these developed after a few moments the child would stand up and grasp the bars of his cot. Then suddenly the breathing would cease altogether and a long apnœic pause of an average duration of about thirty or forty seconds would ensue. In the longest attacks of apnœa the child became very cyanosed, unconscious and flaccid. Finally respiration would begin and he would cry as though frightened, while the cyanosis disappeared.

The attacks occurred, as a rule, at night, but there were occasional attacks by day, if he was excited or emotional. In hospital they varied from two or three a day to twenty or more. On some days there was complete freedom. Physical examination showed nothing abnormal.



The uppermost tracing shows a period of hyperpnœa, with a long apnœic pause before and after.

The middle tracing was taken during a period of normal respiration.

The lowest tracing shows an irregular series of short pauses and hurried respiration.<sup>1</sup>

### (III) F. J. POYNTON, M.D.

We have brought eight cases here this afternoon. There were fourteen in all, grouped into four classes.

In the first group there were three cases, in which there was apparently complete recovery. That always raises the question as to whether the original diagnosis was correct; but I think we can say that all those three patients were true cases of the disease encephalitis lethargica. Those that recovered recovered rapidly. They were all three here to-day.

In the second group were those who had recovered but still had loss of self-control in some way. It is a most important group, showing either emotionalism, or kleptomania, or some other moral delinquency. In some there were severe headaches also. We had four instances in this group. We cannot lay too much stress on them from the point of view of forensic

<sup>1</sup> I am indebted to Professor Arthur Hall, of Sheffield, for kindly reproducing this tracing.—  
H. C. C.

medicine. It is clear from the four cases shown here, as well as from others, that there is now a class of children, who, as a result of having had this disease, are liable to immorality of various kinds.

In the third group we showed two cases. In these, in addition to mental impairment, there were curious choreiform or athetoid movements, which sometimes looked almost epileptiform.

The cases in the last group, of which we showed one example, were those of the Parkinsonian type. The case we showed is like one of advanced paralysis agitans: there are tremor, rigidity, a toneless voice, slow speech, masked face, salivation, some increase in the reflexes, great mental deterioration. All these cases, of which there were five, are in asylums.

#### (IV) E. A. COCKAYNE, M.D.

Patient, a girl, D. B., aged 12 years. In March, 1922, she had a severe attack of encephalitis lethargica with double ptosis, squint, left facial paresis and loss of power in all four limbs. Cerebro-spinal fluid sterile and with no excess of lymphocytes. During her stay in hospital she developed a rapid rhythmical twitching of the left side of the face, which still persists. The rate becomes quicker when she is tired or asleep. She is bad-tempered, irritable and upset about trifles and often goes to sleep in school, whereas before her illness she was a very placid child. Her face is gradually becoming more expressionless.

#### (V) C. P. SYMONDS, M.D.

The case I am showing was reported by my house physician at the time in the *Journal of Neurology and Psycho-pathology*, 1921, p. 249. We could not get any very clear history of the beginning of the illness. The mother states that at first the boy became abnormally drowsy, and that afterwards twitching movements developed, for which he was taken to an infirmary, and admitted with the diagnosis of chorea. He was in that institution six months. When he came out the mother at once noticed the posture of his head, and when she got him home he had nocturnal restlessness. When he came under my observation two years ago he presented, in embryo, the clinical picture he now exhibits; that is, he had some beginnings of the Parkinsonian mask which he shows so plainly to-day. But, as evidence of the fact that the condition has progressed since then, I may mention that I showed him at the Clinical Section a year or two ago, and I had some difficulty in persuading anybody that he had the Parkinsonian aspect. His phase of nocturnal restlessness lasted for about a year, and then completely passed off, and there supervened mental changes of such a nature that he had to be sent to Colney Hatch Mental Hospital, where he still remains. He has violent attacks of temper; when he is thwarted he lies down and kicks, and uses bad language. When at home he could not be controlled. He now sleeps well.

#### (VI) C. WORSTER-DROUGHT, M.D.

*Parkinsonian Syndrome, mainly Unilateral; Sequela of Encephalitis Lethargica.*—B. B., aged 12 years. Onset occurred one evening in June, 1920, with twitching of the mouth and left leg; this was followed a few hours later by drooping of the eyelids and lassitude. He was then admitted to hospital.



On admission (June 10, 1920) the boy was in a profoundly lethargic state and exhibited katatonica and bilateral ptosis. The pupils reacted sluggishly to light, the optic discs were normal, upper limb and abdominal reflexes unobtainable, knee- and ankle-jerks brisk and equal, and the plantar reflexes flexor. The cerebro-spinal fluid showed two lymphocytes per cubic millimetre; weak positive globular reaction (Noguchi); negative Wassermann reaction and a good reduction of Fehling's solution. The patient continued very lethargic and difficult to rouse until June 23; he then developed a tremor of the left arm and occasional clenching of the left hand, associated with transient loss of consciousness (attacks up to three or four times daily).

On July 10, 1920, the spasmodic seizures having disappeared, he was still drowsy, but obeyed commands such as "put out your tongue," &c. Although he did not speak spontaneously, he answered questions regarding his name, age, &c., in a slow and monotonous voice. There was still a tendency to bilateral ptosis, that on the left being more pronounced than that on the right. Facial movements practically absent; tongue very tremulous and on protrusion deviated to the left. The limbs showed general muscular rigidity with an occasional tremor of left arm. All arm-jerks were slight, but equal on the two sides; no inco-ordination of the upper limbs nor intention tremor; right abdominal reflex sluggish, and left absent, knee and ankle-jerks brisk and equal and plantar reflexes flexor.

For about eighteen months he steadily improved, but for the past twelve months his condition appears slowly to have deteriorated. His present state is as follows: He takes an interest in things generally, and although very slow in replying to questions he appears quite intelligent, excepting that at times he is somewhat fatuous and unduly inquisitive. His mother states that he has lost a certain "obstinacy" displayed before his illness. He walks in a rigid fashion, the body being bent forwards from the hips, and the left arm being held immobile, semi-prone and slightly flexed at the elbow with an occasional tremor. There is a tendency to drag the left leg. Propulsion can be demonstrated but not retropulsion. Voice monotonous and slow. Pupils dilated but react normally to light and accommodation; fundi normal; no nystagmus, but slight weakness of right external rectus muscle. Face expressionless with considerable muscular weakness, more marked on right side. The remaining cranial nerves normal. Sensation normal. Arm-jerks moderate and equal on the two sides. Abdominal reflexes sluggish, right being slightly brisker than left. Left knee- and ankle-jerks brisker than those on the right; no ankle clonus; left foot exhibits some degree of flexor contracture of toes, with tendency to pes cavus; right plantar reflex flexor and left unobtainable.

### (B) Discussion.

#### DR. ERIC PRITCHARD (President)

said he had looked up certain of the literature of the subject to see what proportion of cases, according to different authorities, escaped without any after-effects. Some of the writers appeared to be very pessimistic on this matter; one Scandinavian author—S. Tschudi Madsen (*Medicinsk Revue*, October, 1922)—said in practically all cases something abnormal was left behind. It might be that in different countries the after-effects varied.

He had seen in an article in the *Indiana Journal of Medicine* for November, 1922, by W. A. Faulkner, giving the results of 300 cases, from which it appeared that about 25 per cent. of the cases ended fatally, about 25 per cent.

had insignificant after-effects, and about 50 per cent. had serious after-effects. He did not know whether considerable collections of cases in this country showed any correspondence with those findings; but it must be realized by the older members that in the past many cases had not been diagnosed. In 1896 a medical friend of his had two small boys, both of whom were stricken at the same time with what was supposed to be tuberculous meningitis. As both recovered, the diagnosis had to be reconsidered, but no definite opinion was arrived at. One of those boys grew up, and was killed in the war; the other was still alive, but had not fulfilled the promise he gave as a young child; there was something odd and abnormal about him. Both of these boys were probably cases of encephalitis. Another case was that of a small boy in his own family. Up to 3 years of age he had been particularly bright; then he suddenly had an attack of what was thought to be influenza of the cerebral type. He recovered from that, but afterwards his brightness disappeared; he became morose and peculiar, and still remained so.

He remembered also one or two other cases which, in the light of knowledge now possessed, he would have diagnosed as mild cases of encephalitis lethargica.

But there were cases in babies in which it was almost impossible to make a diagnosis. Dr. Cameron had remarked to him that sleepiness in a baby did not count for much; in fact it was considered a favourable sign of well-being. Yet the after-history of some of these babies was very suggestive of the disease under consideration.

#### Professor ARTHUR HALL (Sheffield)

remarked that he had not seen, among the patients exhibited that day, one who was unduly fat; had other members seen obesity in connexion with an outbreak of this disease?

The clinical state exhibited by these children was also seen in adults, though somewhat differently balanced.

He had been much interested in Dr. Cameron's case showing respiratory changes. A case of that kind was recorded in a medical man some time ago; the patient suffering from periodic attacks of hyperpnœa and then long periods of apnœa.

The after-effects of encephalitis lethargica, not only in children but also in adults, were appalling. The more one followed up these cases through a series of years, the more one saw the development of sequelæ, and this development apparently went on for a very long time.

The first case he ever saw was that of a boy 15 years of age, who apparently had myasthenia gravis; he could not move in his bed, he was only able to use his hands. That was in 1918, and he saw the boy again five years later, when he seemed to have recovered well, but had been left with spasm of the face and mouth, with a sudden dropping of the jaw every few seconds.

Restlessness at night was a very common and disturbing symptom in these cases, and the moral effects of the disease were very serious. One boy, aged 14 years, who had previously been a good scholar and of excellent character, suffered from encephalitis lethargica. All it seemed to leave behind was a typical mask face; below the neck he was normal, and his intelligence still remained quite good. His moral character, however, had changed completely. A post was found for him, but after a month he had to be dismissed as he behaved strangely and stole little things; he also became abnormally sexual.

The prognosis in these cases was more serious in regard to after-results than most members of the profession were apt to think.

## Dr. F. PARKES WEBER

said that when Parkinsonian symptoms had supervened on encephalitis, special care should be taken in excluding mental abnormality; in many cases the report was that children after lethargic encephalitis were mentally active and normal. Certainly no such report should be made on a mere hasty examination; it was particularly difficult to ascertain correctly the mental state in the presence of the Parkinsonian syndrome. If great care were not taken, when the patient, apparently recovered, was given some work to do, there might be a disagreeable surprise in store.

One case (male, young adult) seen by Dr. Weber, in which the Parkinsonian syndrome was present, was an extreme example of delayed movement, especially noticeable when the patient yawned. So slow were the movements that they reminded one of the slow-movement pictures shown at cinemas for the purpose of analysing certain movements, such as of jumping or flying. The expression "chameleon-like" had been suggested to Dr. Weber to characterize the movements in question. This was known as "bradykinesia." That patient also had a curious lower jaw tremor. The facial or jaw movements following lethargic encephalitis appeared to be rhythmical tremors but the rate varied a good deal at different times and in different cases; it might vary between 80 and 100 per minute; it might be less frequent.

In another case (male adult) with the Parkinsonian syndrome after encephalitis, when the patient was questioned, he answered not slowly, but very abruptly and harshly, so that the answer appeared to be curt, rude, or resentful. He (Dr. Weber) thought the reason was that in certain cases, as a result of encephalitis lethargica, the higher automatic co-ordination concerned in voluntary movements was especially affected. Normal individuals were accustomed to a flowing movement in answering questions, which was dependent on a kind of spontaneous automatism. When this automatic helping mechanism was damaged by disease in the brain, there was a check in doing what was sought, and this called up a more powerful effort of the will than was normally required for accomplishment. As a result of this altered mechanism, the accomplishment might be somewhat harsh and "explosive,"<sup>1</sup> the defect in the higher automatism having been partially compensated by conscious volitional effort.

## Dr. F. C. SHRUBSALL

said that he approached the subject from the somewhat different aspect of the schools service. The medical officers in the London schools service were attempting to make a card index of cases of encephalitis so as to follow up the children throughout their school career by means of annual inquiries. Some fifty of these cases had come to notice, and further reports had been received with regard to some twenty-five of these. The children attracted attention in school usually either owing to unusual sleepiness in school, to a falling off of the educational attainments, or to a marked irritability or disturbance of conduct.

So far as a general description could be given of cases of such varied type, they might be said to show a combination of apathy with momentary irritability and with some failure of moral inhibitions. A marked feature was the

<sup>1</sup> Compare Verger and Hesnard, "On the mental state of patients with bradykinesia of encephalitic origin," abstract in *Presse médicale*, Paris, 1922, xxx, p. 991.

rapid onset of fatigue and inattention; the children did well at tests which could be done rapidly, but failed at those which required more prolonged planning and concentration, or much exercise of self-criticism. The intellectual impairment seemed to depend largely on lack of desire or interest. The majority of the cases showed a heightened sensibility to environmental stresses, a greater tendency to introspection and a heightened degree of suggestibility, so that in some instances there were definite neurotic symptoms.

Many cases might be said to have made favourable progress towards recovery; the order being first of all an improvement in the physical conditions, the squint and diplopia disappearing within from one to two years of the onset, then a recovery in intellectual activities spread over a slightly longer period, and lastly, often much later and slower, a recovery in the conduct. It was to be noted that misconduct had shown itself chiefly in violent behaviour and wilful damage, in some instances as petty pilfering; there had been no examples of sexual delinquencies.

The clash between an irritable child of somewhat introverted mentality and the stresses of environment readily gave rise to neurotic symptoms, which in many instances might amount to a definite conversion hysteria. As a defensive action against something he disliked (not infrequently school attendance), the child exaggerated already existing symptoms; for example, a child with slight paresis would appear for a time completely paralysed; slight choreiform, athetotic or myoclonic movements greatly increased in amplitude and frequency, dying away again when the subject either got his own way or found this reaction useless for his purpose. Of course these cases would have to be distinguished from the examples in which there was a definite and progressive increase in the physical signs such as had been noted to set in even as much as two years after the original illness. Whether the nervous reaction showed itself in movements, paralysis or misconduct, there was no doubt that the condition was injuriously affected by the reaction between an irritable child and nervous parents who at one moment were foolishly indulgent and at another querulously inflicted partially undeserved punishment. Good results had been obtained by transfer to a residential school or by boarding out away from their home. While under observation, some of these cases had entirely cleared up; others had materially improved. The general experience had been that the misconduct and physical signs rarely coincided, the conduct cases showing the least physical damage, and often little intellectual impairment.

In the majority of the twenty-five cases of which most was known, the initial illness had been characterized by fever and lethargy of varying degree. In three cases there had been symptoms difficult to distinguish from meningitis, in three the lethargy had never been very marked, but there had been some degree of diplopia and paralysis of the facial muscles; in one there had been marked hemiplegia; in two a spastic diplegia, and in another facial paralysis. Two cases had shown marked choreiform movements, two frequent tremors of the hand, one had presented a head-nodding tic, and one definite myoclonus.

The later reports on these cases, after two to three years' observation, were as follows: eight still showed physical disabilities, three in the form of paralysis, four in the form of a tremor or choreiform movements, and one in the form of blindness, due to optic atrophy; two others showed visual defect, one with some degree of double optic neuritis. So far as the mental

condition was concerned, there was little or no intellectual impairment in five, and marked intellectual impairment in eight. On the temperamental side, irritability with outbursts of temper was to be noted in thirteen cases, marked restlessness in five, and no special features in seven. The lethargy had cleared up in all the cases, but five had been reported from school to tend to sleep, particularly over uninteresting lessons. This condition was reported as improving on the occasion of subsequent visits. The effects appeared the more severe the younger the child at the time of the attack. One patient had suffered from encephalitis in the second year of life; previous to this he had been a bright baby, but after his illness became dirty and vicious, with a tendency to coprophagia. When seen at the age of  $4\frac{1}{2}$  years, his mental age on tests was not more than  $2\frac{1}{2}$  years, and his conduct was such that he had had to be transferred to an institution for the mentally defective, whence, however, he is reported as making slight improvement. Two other cases who had had encephalitis during the course of their fifth year, had made definite improvement, although at first there had been marked sleepiness in school and great difficulty in getting to sleep at night, and increased irritability and troublesomeness. The prognosis was not therefore invariably hopeless, at any rate as regards immediate consequences.

A case which showed the benefit of residential school treatment was that of W. J., born 1906, who had suffered from encephalitis with meningeal symptoms in 1918. He came to notice in February, 1919, owing to persistent misconduct in school, violent habits, and frequent complaints of headache. He had previously been in Standard V. The head master at the school had reported that his conduct at the school was usually good, but that his work was very variable, that he failed particularly in arithmetic, and that latterly he had become restless and fidgety, and disliked ordinary school work. On examination at the age of 12 years, it had been found that his response to mental tests was ready and distinctly in advance of his age. A little later there had been evidence that this boy's conduct had become more trying and violent, that he had taken to stealing, and that neither punishment nor remonstrance had any effect. As a result of subsequent thefts he had been sent to an industrial school, where he proved at first very emotional, persisted in minor misconduct, and showed an entirely unrestrained emotional temperament. Under steady discipline he had improved for a time, but on being taken fruit-picking with the rest of the school, he had conceived a dislike to this work and had indulged in threats of suicide. When again seen there had been no sign of intellectual impairment, but a certain degree of emotional instability. His retention at the school had been advised, and in the course of time he steadily improved, and is now in an Army band school, doing well, though he was at times somewhat difficult to handle.

Another case was showing benefit derived from change of surroundings. A. A. (born in 1910) suffered from encephalitis of an abortive type in July, 1920. Previous to this he had behaved well at school and gained a prize, but after his illness he had proved entirely intractable. His physical condition showed a slight degree of right-sided paralysis, but on being laughed at by his relatives and possibly by his school companions, he had become very moody, irritable, and had developed a marked right hemiplegia—an example of a functional addition to a pre-existing organic lesion. Ordinarily, this limb appeared completely spastic, but at times he had been noted to use it, in which case there was a marked intention tremor. When examined at the age of 10 years, he had shown a mental age on the Binet-Simon scale

of 10 years, and on the Healy tests of rather better than 11 years, so that there was no intellectual impairment. His conduct had steadily increased in difficulty of management and violence. There had been obvious conflict with his family and home generally. He had been sent away to fresh surroundings, and was said to be improving steadily.

In another case, M. E. (1907), attacked by encephalitis 1920, there had been marked restlessness, diplopia, and for a time choreiform movements; restlessness at night, sleepiness in the daytime, together with a marked deterioration in attainments. On examination, it had been evident that his attention readily flagged, his mental age on Binet-Simon tests was 11 years, and on Porteous tests 14 years, but his educational attainments were only equal to Standard IV, which was much below his previous standard. In his case, restlessness and troublesomeness had remained a marked feature, and there was some evidence of mental conflict but not of misconduct of a reprehensible character. Some improvement had occurred during the period which had elapsed since his illness, the gain being more marked on the physical and intellectual side than in the powers of inhibition. He had been sent to the country, and reports after a year stated that he was much better, sleeping at nights and being much more normal in his habits. A further report after a second year stated that favourable progress continued and that his conduct was now good.

#### Dr. H. C. CAMERON

said he could support the statement of Dr. Shrubsall that the abnormality of conduct in these children did not differ in type from the abnormality found as a response to faulty parental management. He felt that the conduct of these children was similar to that of the ordinary child who wished to make a sensation and to attract attention to itself.

#### Dr. A. SALUSBURY MACNALT

said that the point raised by the President as to the different sequelæ seen in various countries was one of importance. In their investigations at the Ministry of Health and in their trying to follow out the cases of the disease which had been notified, they failed to find one case which showed the characteristic Parkinsonian syndrome. The cases showed mental changes, and choreiform movements, tremors, &c. His colleagues also had that impression. But at neurological hospitals it was said that the Parkinsonian syndrome in both children and adults was marked, and the cases numerous. In a recent book, incorporating the experience of Professor Pierre Marie and Dr. Gabrielle Lévy at the Salpêtrière, seventy cases out of 129 were said to have the Parkinsonian syndrome. It might be true that many cases of encephalitis in this country were not notified, but, alternatively, the difference might be due to the nationality. It would be interesting to know whether the Scandinavian statistics, dealing with a more phlegmatic people, gave more Parkinsonian cases or less than the Latin and more excitable races.

#### Dr. C. P. SYMONDS

said he was very interested in the discrepancy between the symptomatology shown by the cases which had been notified and which Dr. MacNalty had been able to follow up, and the experiences at neurological clinics. His own



experience at a neurological department had been that, of the cases which came up presenting sequelæ of encephalitis lethargica, only a small minority had ever been diagnosed as suffering from that disease. He believed that this was due to two possibilities: (1) That the initial illness might have been of a very mild degree, and the sequelæ might have been out of all proportion to the initial illness. Recently he had come across two cases in which the sequelæ were very grave; both were Parkinsonian, and both had grave mental deterioration. Neither of these patients had been to bed during the first illness. (2) That the latent period which might elapse between the initial mild illness and the development of sequelæ might be a very extensive one. In the majority of the cases he had seen, the average latent period—when there had been one—between the initial illness and the onset of the sequelæ, had been from three to six months. But he had recently had a most remarkable instance. A boy aged 14 years was brought to his out-patient department because of nocturnal restlessness and wanton destructiveness. He insisted on getting up at four o'clock in the morning and singing so loudly as to wake the household, and that made him think the boy was in a post-encephalitic condition. Inquiry showed that a year previously he had had unusual drowsiness; his friends had consulted a doctor about him, and he had been in bed for a day or two. He had been seen again a month ago, and he was now a typical Parkinsonian case. This did not develop until two years after the original illness, the longest period the speaker had known.

The nature of the underlying pathological process in the cases having the Parkinsonian syndrome seemed to be obscure. There seemed to be two possibilities: (1) That the virus was smouldering the whole time, latent in the nervous tissues. That hypothesis had been supported by experimental work in France, and by the fact that he had seen relapses of the disease, with pyrexia during the relapse. In one case, through the kindness of Dr. J. R. Perdrau, of Lambeth Infirmary, he had been able to examine sections of the nervous tissues after the illness had lasted three years, the initial illness having been only slight. The patient was a woman aged 38 years. Sections through the corpus striatum showed some peri-vascular cuffing; also a marked hyaline degeneration of small arterioles, in some instances going on to calcification. That supported the other hypothesis originally put forward by Dr. Buzzard to explain the progressive nature of these sequelæ, namely, that they were due to a progressive degeneration and to obliterative changes in the vessels, with consequent anæmia of the nervous tissues.

#### Dr. D. McALPINE,

discussing the latent period between the onset of the illness and the onset of the Parkinsonian syndrome, said he had seen over thirty cases. In ten of these there was a latent period of at least six months. The periods varied, the longest periods being between three years and nine months.

The lesion, in the idiopathic form, was considered by Vogt, Ramsay Hunt, and Lhermitte, to lie in the globus pallidus. Tretiakoff, in eight cases, had found the main lesion, on the other hand, in the substantia nigra. Very few autopsy findings had been reported so far in cases of post-encephalitic paralysis agitans; in all of these the substantia nigra was the region most affected. He (the speaker) was at present working on a case in which the patient had died eighteen months after the acute attack, and it was already clear that the main incidence of the disease had fallen on the substantia nigra, though there were also changes in the corpus striatum.



## Dr. BELLINGHAM SMITH

asked whether Dr. McAlpine could say how the pathological changes found explained the extraordinary condition of moral depravity and mental regression which took place in these cases; whether the changes were entirely confined to the substantia nigra or to the basal ganglia, or whether changes had been noted in psychical areas also.

## Dr. McALPINE (in reply to Dr. Bellingham Smith)

said he did not know of any pathology in cases showing changes in moral character. The particular case referred to above showed no mental abnormality, though cerebration was very slow. He had had one case in which dementia præcox definitely followed an attack of encephalitis lethargica; he felt that in such cases the main incidence of the disease was on the cerebrum. Soon there might be opportunities of ascertaining exactly where the lesion was; it might be such as to account for the moral delinquency as well as for the dementia præcox.

## Dr. MACNALT

asked whether any Members had seen the cases of disseminated sclerosis described by Netter, Souques, and others, in Paris, attributed to encephalitis lethargica.

## Dr. C. P. SYMONDS (in reply to Dr. MacNalty)

said he had seen a case at the bedside which was supposed to be encephalitis lethargica, but a careful examination in the light of the history showed it to be disseminated sclerosis; it ran a temperature and looked much like the disease under discussion.

## Section for the Study of Disease in Children.<sup>1</sup>

President—Dr. ERIC PRITCHARD.

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### CORRIGENDUM.

The tracing illustrating Dr. Cameron's case of Hyperpncea with Apnœic Pauses in the last number of the *Proceedings*, No. 6, April (Sect. Study Dis. Child., p. 31) was reproduced upside down and should be inverted.

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### Case of Congenital Syphilis.

By J. H. THURSFIELD, M.D.

PATIENT, a male, aged 13 months. At 11 weeks of age he had iritis and keratitis, with consequent destruction of vision of eye. Sir Jonathan Hutchinson described this rare condition and pointed out that it is nearly always unilateral, as in this case, and more often found in girl than in boy babies: his figures are twelve girls to three boys. The patient also had well-marked epiphysitis.

Dr. J. H. THURSFIELD showed a Girl, aged  $4\frac{1}{2}$  years, with Polyarthrititis, present since the first year of age.

### Two Cases of Acholuric Jaundice.

By ROBERT HUTCHISON, M.D.

PATIENTS, a girl, aged 11 years, and a boy, aged 6 years. Both children have had recurrent attacks of jaundice with increased fragility of red-blood corpuscles and enlarged spleens. Splenectomy has been performed in both cases by Mr. Addison. Good recovery. No recurrence of attacks. Fragility of red blood corpuscles still remains.

### Case of Acholuric Jaundice.

By DONALD PATERSON, M.B.

PATIENT, a girl, aged 9 years. There is a large spleen, increased fragility of red blood corpuscles, and the jaundice is worse at times.

<sup>1</sup> Clinical Meeting at the Great Ormond Street Hospital for Sick Children, held February 23, 1923.

### Specimen of Congenital Stricture of the Œsophagus.

By ROBERT HUTCHISON, M.D.

THE patient was a boy aged 3 years. Since he was 6 weeks old he had had attacks of vomiting and constipation about once a month. The attack lasted about seven days and during this time everything was vomited. In between these attacks he usually vomited about once a day. Was always constipated. The vomited matter is bile-stained during the attacks. On admission to hospital the attack had lasted nineteen days. Past history: Had never taken solid food. Walked at the age of 1 year 9 months. Family history: Four healthy children; patient being the youngest. Was extremely constipated on admission and gave the picture of acidosis. Vomiting continued, although the acidosis had been adequately treated. A bismuth meal revealed the stenosis of the Œsophagus. The child wasted rapidly and died six weeks after admission.

*Autopsy.*—Stricture of Œsophagus  $1\frac{1}{2}$  in. below bifurcation of trachea. Œsophagus dilated above the stricture, probe could not be passed, water trickled slowly through. There was no sign of past inflammation or enlarged glands. The stricture was hard and symmetrical, like a small hard fibrous pylorus.

### Case of Progeria.

By DONALD PATERSON, M.B.

PATIENT, a boy, aged 8 years. Condition has been present since birth. The father and mother are first cousins. There are four children in the family: the girls are unaffected, both boys are affected. The senile condition of the skin and facies should be noted. The vessels show arterio-sclerosis. There is almost complete absence of subcutaneous fat.

Dr. DONALD PATERSON also showed Two Cases illustrating the Schick Test.

### Cases of Congenital Syphilis.

By D. NABARRO, M.D.

*Case I.*—Female, aged 17 months. Congenital syphilitic child of a congenital syphilitic father (Wassermann reaction ++++). Mother's Wassermann reaction negative even after a provocative injection of novarsenobillon. The child was only mildly infected with symptoms of convulsions, wasting and rash. Wassermann reaction +++- (August 16, 1921). Negative three months later, and since. Treatment: Mercury for fourteen months. Paternal grandmother—Wassermann reaction ++++.

*Case II.*—Patient, a boy, aged 8 years. Hemiplegia. Enlarged head. Knee swollen July 10 to August 31,<sup>1</sup> 1921, and again in March, 1922. Interstitial keratitis (February, 1922).

Had nine months' treatment with mercury and iodides in 1919-20. Injections since January, 1922. Wassermann reaction ++++ and also in cerebro-spinal fluid.

*Case III.*—Patient, a girl, aged 12 years. Deafness, keratitis, ulceration of palate (now has a cleft). Has had forty-six injections totalling 16·6 grm., but Wassermann reaction still + + + +.

*Case IV.*—Patient, a boy, aged 10 years (brother of following patient). Ear discharge and nasal ulceration. Symptoms very similar to those seen in the sister. Patient has had forty-six injections totalling 13·3 grm. Salvarsan substitutes, but Wassermann reaction is still + + + +.

*Case V.*—Patient, a girl, aged 13 years. Had condyloma when 1 year old. Five years' treatment with mercury. Eyes: Choroiditis (1914) at the age of 5 years. Interstitial keratitis. Typical Hutchinsonian teeth. Injections of novarsenobillon begun November, 1922, with definite improvement in vision resulting. Mother and three younger children all give negative Wassermann reactions.

*Case VI.*—Patient, a girl, aged 13 years. Otorrhœa and ulceration of palate, keratitis. Patient has had forty injections totalling 13·3 grm., but the Wassermann reaction is still + + + +.

Dr. F. J. POYNTON showed a Case of Cretinism.

### Museum Specimens.

Demonstrated by C. B. DANSIE, L.R.C.P., M.R.C.S.

(1) *Still's Disease.*—Joints showing extensive erosion of articular cartilage. Heart showing adherent pericardium, soft adhesions which could be easily separated—an exudate rather than adhesion between the visceral and parietal pericardium.

(2) *Sclerosis of the Brain.*—Infant, aged 6 months. Progressive weakness of limbs and mental deterioration. Fundi normal.

(3) *Congenital Honeycomb Lung.*—Boy, aged 6 years 8 months.

(4) *Tuberculous Spleen.*—Infant, aged 9 weeks.

(5) *Hip-joint from a case of Lymphatic Leukæmia.*—Child, aged 2½ years.

### Exhibition of Cases.

By H. TYRRELL GRAY, M.Ch.

(1) *Congenital Absence of Shoulder Muscles, Right Side.*

(2) *Congenital Absence of Radius with Extreme Eversion of Hand.*—Treatment by bone-graft at age of 3 years, and removal of thumb at the age of 6½ years.

(3) *Congenital Hydrocephalus in a Girl.*—Drainage of ventricles with puncture of corpus callosum at age of 8 months. Patient now aged 6 years. Mental condition good.

(4) *Congenital Hydrocephalus in a Boy.*—Drainage of ventricles with puncture of corpus callosum at age of 10 months. Now aged 4 years.

(5) *Acute Osteomyelitis of Right Tibia in a Girl aged 8 Years.*—Ununited fracture treated by successive bone-grafts.

(6) *Congenital Absence of Pectoral Muscles in a Male Infant aged 16 months.*

(7) *Dextrocardia without Transposition of Other Viscera.*

### Case of Ankylosis of Jaw.

By G. E. WAUGH, F.R.C.S., and A. T. PITTS, D.S.O.,  
L.R.C.P., M.R.C.S.

PATIENT, a girl, aged 9 years. History: Abscess in front of right ear when 8 months old. Difficulty in opening jaw since. In 1917, when aged 4 years, operation by Mr. Waugh: jaw open  $\frac{3}{4}$  in.; gags under anæsthesia; props and wedges at night. Improvement. In 1918-20: Similar treatment. In 1921, aged 9 years: Could only separate teeth  $\frac{1}{2}$  in.; incisors of lower jaw behind upper, and lower premolars and molars inner side of upper jaw; incisors did not meet. Operation by Mr. Waugh: Forcible gagging under anæsthesia; props; gland abscess in neck opened.

May 12, 1922, operation by Mr. Waugh: Excision of right temporo-mandibular joint; no joint cavity found; condyle hewn out of solid mass of bone: dental impression for splint, taken by Mr. Pitts. Movements improved.

*The Result of the Operation.*—An impression of the jaws was taken at the time of operation after the condyle had been excised. Metal cap splints covering the crowns of the teeth were made. These had extensions coming outside the mouth on each side; those on the lower splint projected upwards, and those on the upper splint projected downwards. The splints were cemented on the teeth, and elastics were attached to each pair of extensions. The pull of the elastics tended to keep the mouth open, and it could only be shut by overcoming the resistance of the elastic. These splints were kept on for several weeks and then removed. The patient has now been free from any apparatus for some considerable time. She has a good functional bite, which has not shown any tendency to lessen by fibrous contraction at the site of operation.

### Congenital Absence of Teeth in Three Members of a Family.

By A. T. PITTS, D.S.O., L.R.C.P., M.R.C.S.

THE patients, two boys, S. A. and J. A., both have two peg-shaped teeth in the lower jaw, next to the canines, with a space between them. The mother states that there were no deciduous predecessors, and that these teeth have not been lost, although the other permanent incisors have erupted. She states that a twin brother of J. A., now dead, had a similar condition. According to this history, which would appear to be correct, the abnormality consists of an absence of both deciduous and permanent central incisors with an absence of the permanent lateral incisors. The deciduous lateral incisors are peg-shaped. This is distinctly rare, especially in the lower jaw. Absence of deciduous teeth is uncommon, although the opposite condition of supernumerary deciduous teeth is often seen. Peg-shaped teeth usually occur in the upper lateral incisor

region, the upper laterals being the teeth which most commonly show abnormality of shape, or they may be entirely absent. The upper third molars are also frequently reduced in size, though, perhaps, less commonly absent. In both cases the explanation has been suggested that owing to the reduction in size of the jaws there is a gradual tendency towards suppression of the lateral incisors and third molars in the upper jaw. The hereditary factor in these two cases is a feature of interest. I could not get any evidence of inheritance. In another case recently seen here, that of a mother and her two daughters, there was absence of upper laterals; in the mother it was unilateral, in the daughters it was bilateral, while in one of them a lower incisor was absent.

### **Case of "Hutchinsonian" Teeth.**

By A. T. PITTS, D.S.O., L.R.C.P., M.R.C.S.

THE upper central incisors are peg-shaped, and show a marked narrowing toward the cutting edge. Wassermann reaction +++. It is a mistake to assume that the presence of a notch is invariably present in congenital syphilis, though this type undoubtedly presents the "Hutchinsonian" tooth in its most perfect form. In all cases the tooth shows a narrowing toward the cutting edge, and unless this is present I should hesitate in regarding any tooth as of the "Hutchinsonian" type. The cutting edge, however, shows a range of variation. In the least marked form the edge may be normal, and there is not any bevelling of the enamel adjacent to the cutting edge. In the next type there is a crescentic area of bevelled enamel just above the cutting edge, but the cutting edge shows no notching. I would suggest for this area the term, "potential notch." The next type shows a "potential notch," but in the centre of the cutting edge there is a small deficiency—an actual notch. In the next type there is a definite crescentic notch in the cutting edge, with a smaller zone of "potential notch" above it. This last represents the Hutchinsonian tooth as Hutchinson himself described it, and as it is always described by later writers. The suggestion has been made that the notch only develops through actual attrition of the cutting edge, which wears away the badly formed enamel of the potential notch. This may be so in some cases, though I have not been able to obtain proof of it. It is, however, definitely incorrect as a complete explanation, for it is usual to see teeth just erupted which show a well-marked notch. Indeed it may be present before eruption, and can be demonstrated by the X-ray, as shown in the skiagram now exhibited. In this case one central incisor had erupted and showed a well-marked notch. The other had not erupted, but the X-ray shows it in the bone with a definite notch.

The sister of this child is a typical case of congenital syphilis.

### **Congenital Absence of all Teeth except Two.**

By A. T. PITTS, D.S.O., L.R.C.P., M.R.C.S.

PATIENT, B. P., aged  $7\frac{1}{2}$  years. The only tooth present is a molar in the left maxilla. The X-rays show another molar present in the right mandible. The boy was one of twins, the other dying at birth. Birth was premature and

the child delicate as a baby. He is very small for his age. The mandible has an obtuse angle like that of an edentulous person. The alveolar ridges and palate are very shallow. The hair is coarse and dry; the cheeks are somewhat scaly. As a baby the skin was said to be normal. This case should be compared with a similar case shown by Dr. Cockayne<sup>1</sup> before this Section about two years ago. Several of these cases have been recorded from time to time. As the lack of teeth is a striking feature they are often described from this point of view. In all cases there seem to have been associated abnormalities of other epithelial structures. In comparative anatomy it is not unusual for epithelial structures to vary together, and this is called "correlation of variation." In the deer the canine teeth are normally absent, but in certain species in which the antlers are absent or rudimentary, e.g., the musk deer, Chinese water deer, and Swinhoe's deer, the canines in the upper jaw are well developed in the male, and present, though small, in the female. This would suggest as a possible cause an internal secretion of the gonads. By analogy such defects, as shown by the case of this boy, might be assumed to be due to some congenital deficiency of one of the endocrine organs. In this connexion I would also refer to L. Bolk's theory of the ductless glands being a factor in evolution. The condition cannot be due to any malnutrition after birth, for the first dipping in of the oral epithelium to form the tooth-germs occurs at about the fortieth day of foetal life. Evidently the primitive ectoderm is at fault. In some cases of this type investigated in Germany it was found that there was an absence of sweat glands, while I am informed by Dr. Cockayne that the hair has been found to be abnormal in these cases. The Wassermann reaction was negative in this case.

Mr. P. G. DOYNE, F.R.C.S., showed: (1) A Girl with Hernial Protrusions of Orbital Fat on either side of right and left Globes, more marked on the outer side. (2) A child with an additional Phalanx in the right Thumb

<sup>1</sup> *Proceedings*, 1920-21, xiv (Sect. Study Dis. Child.), p. 17.



## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

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### Raynaud's Syndrome in a Non-syphilitic Infant, with a Remarkable Family History.

By F. PARKES WEBER, M.D.

(ABSTRACT.)<sup>1</sup>

THE patient, N. M., aged 12 months, of an originally Russian Hebrew family, was admitted to hospital on September 20, 1922, suffering from recurrent attacks of Raynaud's syndrome. During an attack the affected part was at first blue, cold to the touch, and obviously painful. This cyanotic stage of "local asphyxia," as the attack passed off, was succeeded by one of reaction, with active hyperæmia, and during that stage the part became bright red and hot to the touch; then all traces of the attack rapidly and completely disappeared. The attacks could be cut short by the application of a rubber hot-water bottle. The internal use of benzyl-benzoate seemed to diminish the angiospastic condition. The attacks became less severe, shorter and less frequent, but a period of complete freedom in October, 1922, may have been connected with the temporary occurrence of slight febrile bronchitis. The attacks were of the cyanotic type ("local asphyxia"), never of the "dead" white type ("local syncope"). The Wassermann reaction had been elsewhere found to be negative in the patient and in her mother. Between the attacks the child presented no definite signs of disease, excepting adenoids and frequently a muco-purulent discharge from the nostrils.

According to the history given the child was born at full term; the confinement was normal. She was breast-fed for the first six weeks and afterwards partly bottle-fed, partly breast-fed. The Raynaud attacks were first noted at the age of 3 months. There was, as usual in cases of Raynaud's disease, no history of Raynaud's syndrome occurring in other members of the family, but the family history was remarkable. The father, now aged 30 years, known to Dr. Weber since 1911, used to suffer from recurrent attacks of headache and vomiting and formerly had a slightly enlarged spleen; he had a negative Wassermann reaction. A paternal aunt and a paternal uncle had diabetes mellitus and died at the ages of 18 and 20 years respectively. The paternal grandfather, a Russian Jew, now aged 58 years, had been frequently seen by

<sup>1</sup> The paper is published in full in the *British Journal of Children's Diseases*, January-March, 1923, xx, p. 25.

## 48 Weber: *Non-syphilitic Infant*; Kendall: *Ductus Arteriosus*

Dr. Weber since 1906 for thrombo-angiitis obliterans, from which he commenced to suffer at the age of 38 years. His Wassermann reaction was negative.

### DISCUSSION.

Dr. ERIC PRITCHARD (President) asked whether the headaches and sickness—probably migrainous in nature—in the family history might not have been due to arterial spasm; and whether the history of bronchitis might not have indicated asthma with spasm of the bronchial arteries? Had Dr. Weber any explanation, physiological or mechanical, of the difference between the cyanotic type and the syncopal type with pallor?

Dr. JEWESBURY asked whether the urine was affected in this case.

Dr. PARKES WEBER (in reply) said that the urine showed nothing abnormal. With regard to the difference between the local cyanotic attacks and the attacks of local pallor, he thought that the capillaries and venules took part in the angiospasm of the latter ("local syncope"), and that in the cyanotic attacks ("local asphyxia") there was a tendency to dilatation of the capillaries and venules, though the arterioles were contracted. He had never heard of Raynaud's symptom-complex being present in several members of the same family, but, in cases of Raynaud's symptom-complex, other members of the family might, he believed, be affected with asthma, migraine, &c.

## Case of Patent Ductus Arteriosus and Mitral Disease.

By G. M. KENDALL, M.B.

THE patient, a girl, aged 8 years, had scarlet fever at the age of 1½ years and measles at 5 years. She has had no other serious illnesses. Two years ago her nose bled frequently, but has not done so since. The heart was examined in July, 1922, and said to be "weak." The present illness has lasted for four months. She has been languid, and short of breath and has lost her appetite. Pains have been complained of in the arms and legs and in the left side. She had a sore throat about three months ago. She has now been two months in hospital and the condition on physical examination has not altered. The pulse varies from 120 to 140.

The heart is moderately enlarged to the left but not to the right; the upper limit of cardiac dullness extends up to the first interspace close to the sternum. The impulse is forcible. There is a long blowing and partly musical systolic murmur at the apex which is conducted towards the axilla. At the base there is a well marked thrill and murmur and on these signs the diagnosis of patent ductus is based. Both the thrill and murmur are most noticeable in the first and second space near the left border of the sternum. The thrill is felt faintly in the third space also, but not elsewhere. It appears to be intensified at the first and second sounds and to die away during the long pause. The murmur is best described as "roaring" and at its loudest point may be compared to the noise of a train in a tunnel. It is continuous and is intensified at two points which are synchronous with the first and second sounds. Lower down, in the third and fourth spaces near the left sternal edge what appears to be the diastolic component of this murmur is heard, but here it is very superficial and rather like an exocardial sound.

There is no œdema.

The liver edge is felt 2 in. below the right costal margin: there are no rheumatic nodules.

The temperature very occasionally rises to 99° F.

The skiagram of the chest shows the heart enlarged to the left and not to the right: the outline of the aortic arch is obscured, possibly by a dilated pulmonary artery, but this cannot be decided from the appearances.

The blood contains 4,750,000 red cells per cubic millimetre.

The condition appears to be one of patent ductus arteriosus, combined with mitral disease of rheumatic origin.

#### DISCUSSION.

Dr. ERIC PRITCHARD (President) said that there were one or two anomalous points about the case. If there was a serious congenital lesion, the patient was remarkably well grown for 8 years of age. And if the rasping vibration was due to a congenital condition, it seemed strange that so intense a form should not have been recognized long ago, especially by the medical attendant, who, however, had simply remarked that the heart was weak. The whole condition was compatible with a mild myocarditis combined with chronic pericarditis. The sound altered very much on pressure, but that might be due to a patent ductus arteriosus. It was also to be noted that the child seemed in good health until the definitely rheumatic conditions came on. He (the President) questioned the patent ductus arteriosus, though many of the physical signs favoured that view.

Dr. BELLINGHAM SMITH said he thought Dr. Kendall's diagnosis was correct; he regarded the physical signs as quite typical of patent ductus arteriosus. There was the increased band of dullness at the base of the heart, a well marked thrill, and the fairly typical murmur, which varied considerably in different cases; it might be one running through systole and diastole, or the two portions of the murmur might be dissociated, as in this case. There was a marked accentuation of the second sound (pulmonary) and the murmur was propagated in the usual direction, towards the left clavicle. With regard to this patient being well grown for her age, in many cases of congenital heart disease the patients developed and grew normally in stature. He (Dr. Bellingham Smith) had seen two cases of patent ductus arteriosus—both proved post mortem—one in a woman, aged 59 years, the other in a girl, aged 21 years. Neither had seemed to suffer any disability from the congenital lesion until they developed infective endocarditis at the site of the patent ductus arteriosus. The enlargement of the heart was due, he thought, to the recent rheumatic carditis.

Dr. MYERS said he thought there was almost certainly a patent ductus arteriosus here. Apparently no cap has been found above the heart by X-ray examination. The pulmonary second sound was "plus," which was typical of patent ductus arteriosus, but it might also be accounted for by a mitral lesion. He had known several cases of patent ductus arteriosus in people of about 40 years of age, and they were persons in good health.

Dr. F. PARKES WEBER asked whether any Member had ever seen a case of communication between the pulmonary artery and the aorta which was not patent ductus arteriosus. He referred to a communication very near the orifice of both vessels just above the valves. That was said sometimes to occur either congenitally or as a result of traumatism and atheroma, with bulging of the aorta in that region.

Dr. VINCENT COATES said that in this patient the apex beat of the heart was fixed. Change of position showed that there was no mobility of the apex beat. The matrix of the child's finger nails was shiny, a condition he associated with *early* finger clubbing. For these reasons he (Dr. Coates) thought that it was a question of pericarditis and myocarditis in addition to one of patent ductus arteriosus.

Dr. KENDALL (in reply) said he would emphasize the fact that the child had been in hospital two months, during which time the signs at the base of the heart had not altered in any way; and this fact strongly favoured the view that this trouble was of congenital origin. Very likely there might be some adherent pericardium, but he did not think the loud murmur and thrill could be due to pericardial friction.

**Two Cases of Duodenal Obstruction in Infants treated by Operation.**

By R. C. JEWESBURY, M.D., and MAX PAGE, F.R.C.S.

(I) R. C. JEWESBURY, M.D.

At a recent meeting of this Section I reported two cases of duodenal obstruction in infants who had both died. This condition was confirmed by post-mortem examination. I am now reporting two further cases of the same condition which were both operated on by my colleague, Mr. Max Page. The particular interest in these two cases consists in the fact that the condition was relieved successfully by surgical measures. In the literature it is difficult to trace how many of these cases have been dealt with surgically, but apparently very few have been so treated and there appear to be no previous records of cases which have been cured by operation.

*Case I.*—A female child, born at full term, birth-weight 9½ lb. Breast-fed since birth but had forcibly vomited after nearly every feed; the vomit was green in colour. The bowels were constipated. On the eighth day the child weighed 8 lb., i.e., 1½ lb. below the birth-weight and then was obviously wasting. The vomit continued to be forcible and deeply bile-stained. The stomach was dilated but no tumour could be felt and no peristalsis was visible. The stomach was washed out and the breast-feeds were reduced in number but the vomiting continued as before. By the twelfth day the weight had fallen to 6 lb. 12 oz., and the child was admitted to St. Thomas's Hospital. The stomach was washed out, bismuth solution (3 oz.) was given through a tube and an X-ray photograph was taken (fig. 1). This showed a patent pylorus and a duodenal obstruction, which appeared to be almost complete, at the junction of the second and third parts. This showed better on the screen than on the plate. An operation was then decided on and this was performed by Mr. Page when the child was twelve days old. Saline solution was injected subcutaneously before and after the operation. The feeds, after the operation, consisted of very small amounts of diluted breast-milk which was gradually strengthened and increased in amount. Five days after the operation the bowels began to act and the motions were normal. There was occasional slight vomiting for a time after the operation but eventually the child got back to the breast and is now doing exceedingly well. At two months the weight was 8 lb. 4 oz., showing a big gain since the operation.

*Case II.*—A female child aged 5 days; full term birth (weight, 7½ lb.); breast fed. History of forcible vomiting, from birth; the vomit was bile-stained and contained some mucus; the meconium was bile-stained. Nothing abnormal could be felt in the abdomen and there was no visible peristalsis. X-ray examination after a bismuth meal given by tube showed the stomach much dilated and an almost complete obstruction at the duodeno-jejunal flexure (figs. 2, 3). Immediate operation was decided upon and this was done by Mr. Page when the baby was 5 days old. After operation the child was fed in a similar fashion to the former case. She did well for a time and gained weight but after some days bile-stained vomiting returned and the condition appeared to have recurred. She is still in hospital, the stomach remains dilated and is being washed out daily; the wash-out contains bile and mucus. The child is not in a fit state to stand a second laparotomy.

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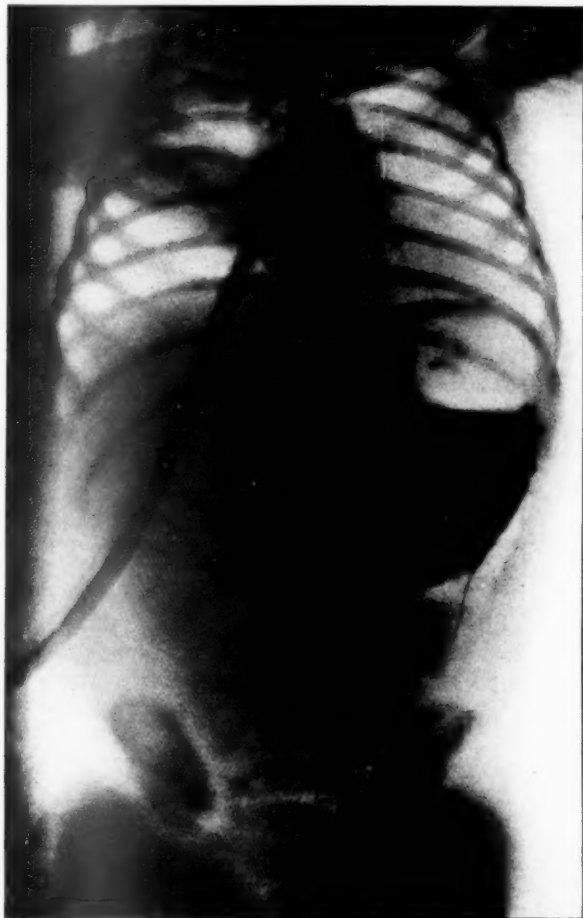


FIG. 1 (Case I).—Showing dilated stomach, pylorus and duodenum, with obstruction at lower part of duodenum. Bismuth meal given by tube.

(II) C. MAX PAGE, D.S.O., M.S.

Dr. Jewesbury has said that the condition found in these cases was the same as he had described on a previous occasion; though this was true of the clinical condition, pathologically the obstruction was produced in a different way.

In the first case, on opening the abdomen the stomach was found to be obviously dilated, as were also the second and third parts of the duodenum. The small bowel was collapsed. The obstruction was clearly at the duodenal flexure, and it appeared to be due to the turning over from left to right of the

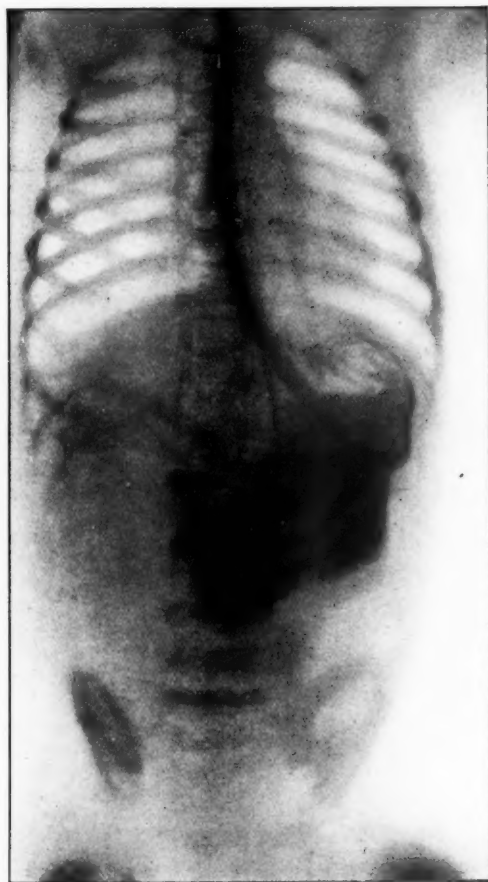


FIG. 2 (Case II).—Showing dilatation of stomach and obstruction in duodenum.

proximal part of the jejunum. The jejunum passed through a segment of mesentery of the small intestine half an inch from the duodeno-jejunal flexure. The obstruction was relieved by freeing the first two or three inches of the jejunum from adhesions which bound them down to the peritoneum on

the posterior abdominal wall and to the upper surface of the mesentery of the small bowel. The adhesions were avascular, and did not require ligaturing.

In the second case the first, second and third parts of the duodenum were dilated, and what was especially striking was the enlargement of the second part of the duodenum. The relationship of the first part of the jejunum to the mesentery was apparently normal, but there was the same tying down of the first part of the jejunum over the right side. The peritoneal attachments of the large bowel were mobile.

With regard to the surgical view, it is clear that in these cases of duodenal obstruction, operation is justified when the condition is promptly diagnosed by X-rays, because if even in only a small proportion of the cases it should prove

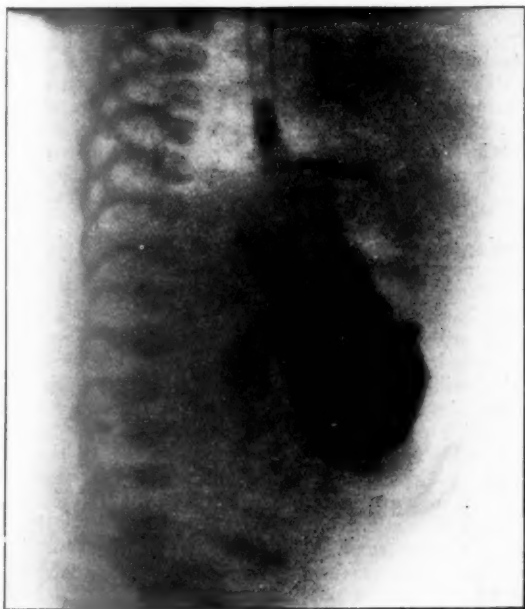


FIG. 3 (Case II).—(Lateral view). Showing bismuth in stomach and upper part of duodenum.

possible to relieve the obstruction by dividing adhesions, the procedure is well worth attempting. According to the literature, most of the cases in which operation has been tried were those of stenosis of the duodenum; in such cases the performance of an anastomosis is necessary, and in the first week of life such a proceeding seldom meets with success.

Dr. ROBERT HUTCHISON said that he did not think these cases were so rare, as would be gathered from the literature; he had himself seen quite a number of them at Great Ormond Street Children's Hospital, and had one under his care now; Mr. Higgins and he had had one recently in which there was obstruction in the



duodenum. These cases presented superficial resemblances to cases of pyloric stenosis, the difference being that in pyloric stenosis the vomiting did not begin from birth. If there was bile-stained vomit, it was also suggestive. Both these features were present in the case referred to. Operation was decided on, and Mr. Higgins did a gastro-enterostomy, although the child was only about a week old and not in good condition. Afterwards there was, for a time, great difficulty in feeding, and constant vomiting, which was checked by giving pancreatic extract. Usually he (Dr. Hutchison) was sceptical as to the value of digestive extracts, but in this case it had made all the difference. Possibly the entrance of pancreatic juice had been interfered with. The child was now a fine baby.

### Case for Diagnosis.

By R. C. JEWESBURY, M.D.

A FEMALE child, aged 3½ years. Normal at birth. Breast-fed eight months. She was quite well until a year ago, when she had a severe attack of broncho-pneumonia, with which she was ill for three months, fever lasting for two months. She has never walked since this attack. Six months ago a "lump" was noticed in front of the right hip, and at the same time swelling occurred in the wrists and elbows. The child has also had irregular fever for the past six months. The joints are not painful except when moved much.

On examination: She is very small for her age. Weight, 21 lb. 2 oz. She is very thin and weak, the skin is dry and shows a light brown diffuse pigmentation. There is an excess of hair all over the body, especially on the forehead and back. The conjunctivæ are pale. Intelligence is normal. Both wrist-joints are distended with fluid, movements are limited but not painful unless forced. Both elbow-joints are somewhat swollen, and full extension is not possible. There is also a little fluid in both shoulder-joints. The small joints of the fingers are slightly swollen, the toes and ankles are not affected. The right hip is ankylosed (bony ankylosis), and the thigh is flexed and much abducted. A hard bony outgrowth is felt in the upper third of the femur, and above this is a mass of enlarged glands, extending into the right groin. The glands are enlarged in both axillæ and in the neck. The teeth are carious and septic. The liver edge is palpable, the spleen is not felt. Lungs and heart normal. The spine is rigid but, on X-ray examination, shows no bony changes. The urine is normal. The blood shows a leucocytosis of 22,000 of the polymorphonuclear type. A blood culture is negative. Fluid withdrawn from a wrist-joint has been cultured—it is sterile and contains no cells. The Wassermann reaction (blood) is negative. A gland removed from the axilla shows a general lymphatic hyperplasia, but there is no evidence of tuberculosis. The temperature is irregular, varying between the normal and 102° F.

An X-ray examination of the wrists shows a synovitis with practically no bony changes, but X-ray examination of the right hip shows a partial bony ankylosis with a large bony outgrowth involving the joint and the upper third of the right femur.

The question is to fit in the condition of the right hip and femur with the other joint changes which, including the hip, would do for a polyarthritides of the type described by Still. In the hip there is a distinct condition which almost suggests a periosteal growth.

## DISCUSSION.

Dr. ERIC PRITCHARD (President) said the clinical picture resembled that of tubercle, and that possibility was not negated because the examination of one gland failed to give a positive result. Probably the inoculation of a guinea-pig with the serum from one of the joints might give an answer to the question. If the condition was septic, there would be more evidence. The only fact in favour of the theory was the excess of polynuclears, such as one would not expect in tuberculosis.

Dr. BELLINGHAM SMITH said he would agree that this was a case of Still's disease: there were pigmentation, multiple arthritis, and enlarged glands. The difficulty appeared to be the bony enlargement in the region of the right hip. He thought it was chronic osteoperiostitis, and if Still's disease was a chronic infective disorder, the same cause might produce osteoperiostitis.

Dr. R. HUTCHISON said that all that could be remarked about this case was, that it was a chronic multiple infective arthritis. A multiple condition such as this was very unlikely to be tuberculous. It might be called Still's disease, because that was not an entity; it might be due to different infective agents. He considered it a bad principle in diagnosis to diagnose two separate conditions if both could be brought under the same pathological cause, so he would be tempted to say that the condition of the right hip in this patient was in some way due to the same pathological process as that in the other joints. He would be reluctant to say that one was dealing here with the infective arthritis called Still's disease plus something different which affected the right femur. Probably they were both the result of the same chronic infection. It might be very difficult to discover the nature of the infection, as he had had fluid from cases of Still's disease frequently cultured, without deriving any help from it. It might be more helpful to examine material from the bladder or from the spleen.

## Case of Tay-Sachs Disease.

By L. MANDEL, M.D.

(Shown by E. BELLINGHAM SMITH, M.D.)

FEMALE infant, aged 20 months.

Family history: Parents are first cousins, both alive and well. Father (aged 27 years), born in London and of pure British parentage; grandparents British. Has never had any specific disease or serious illness. Mother (aged 24 years), of London birth, previous history good. Family antecedents for two generations also non-Jewish. Has child 3 years of age healthy: no miscarriages.

History: Full-term child, normal labour and delivery. Weight at birth, 7 lb. Breast-fed up to age of 10 months, then fed on cow's milk and water.

Brought to my out-patient's department on account of "having a weak back" (could not sit up). There had been difficulty in weaning. The mother had not noticed anything strange about the eyes except that the baby "looked a little vacant." After the seventh month, however, she would lie still in any position in which she was placed and was very quiet during the day but noisy at night.

N.B.—There is a history of the baby having had a fall when 4 months of age; this caused "black eyes" but she soon recovered.

On admission, September 6, 1922: Weight, 11 lb. 10 oz. Tongue not protruding; not badly nourished; looks vacant, takes no notice of surroundings, does not perceive light; is very sensitive to sudden noise which

attracts her attention, otherwise lies quiet and appears only to eat and sleep; gives a strange plaintive cry when hungry—particularly at night when there is no noise. When she is placed in a sitting position her head rolls about. Muscular development is fair; not distinctly spastic. Takes food well. Anterior fontanelle widely open. Nothing abnormal observed in heart, lungs or urinary system; abdominal muscles lax, nothing abnormal felt; no teeth; constipation, knee-jerks brisk; Kernig's sign indefinite; no clonus. Cranial nerves intact.

Eye condition: Mr. A. H. Levy reports: "There appears to be no perception of light. The discs are atrophic, white clear cut edges, vessels normal in size. Both maculae are occupied by white area with circular dark red spot in centre." The case was shown at the meeting of the Section of Ophthalmology on January 12, 1923, and the condition of maculae was considered typical.

Wassermann and von Pirquet reactions negative, cerebro-spinal fluid, normal pressure, cell content normal, Wassermann negative.

Progress (November 16, 1922): Continued to take her food well (cow's milk and water, then Glaxo). Constipation less; general apathy unaltered, spasticity slightly more evident and there was a tendency for the hands to assume a "main en griffe" position. Thyroid extract was given  $\frac{1}{4}$  gr. and "anterior" pituitrin  $\frac{1}{2}$  gr. twice daily. Attempts were made to give slightly more solid food—gruel, &c., but the infant seemed unable to swallow anything but liquids. She gained 2 lb., however, in hospital. Her mother took her home on this date.

January 10, 1923: When seen to-day, aged 17 months, much wasted; weight, 11 lb. 14 oz. (a loss of 2 lb.); spasticity more marked; (unable to sit up unsupported); still unable to take anything but liquid; auditory acuity marked. Now has four teeth appearing. Fontanelle still wide open.

February 11, 1923: Has gained 7 oz.; still unable to sit up. Spasticity variable; according to mother "child is quite stiff at times." Still unable to swallow anything beyond liquids.

The particular interest of the case is that, so far as can be traced, there is no history of Jewish antecedents.

### Case of Enlarged Liver with Persistent Acetonuria and Diaceturia.

By C. WORSTER-DROUGHT, M.D.

A. P., A GIRL, aged 10 years, first seen on June 10, 1920.

Family history: Father and mother both alive; father has always had good health, but mother is deaf (otosclerosis). The mother was the eldest of eight children; of the seven, three died of phthisis, one had a tuberculous wrist-joint, two were epileptic and only one quite healthy. In the father's family there is nothing of note. Wassermann reaction (blood): Father and mother both negative. Patient has a sister (aged 7 years) said to have an "enlarged liver." On investigation of this sister, the liver edge was found to be palpable 2 in. below the costal margin, but X-ray examination indicated ptosis of the organ rather than enlargement; the spleen was impalpable and

there were no other physical signs of importance. Wassermann reaction (blood): negative. The only other child had died twelve years before at the age of 3 years; he was said by a doctor to have an "enlarged liver and spleen," and to have died of "kidney disease."

Previous history: Mother states that pregnancy and labour were normal; at birth the child weighed 11 lb. She was breast-fed and was perfectly well until a year old. From the age of 1 to 4 years, she suffered from "fits" (description suggesting epilepsy, with average frequency of one fit in two months), and between the ages of 3 and 5 years suffered from recurrent



attacks of vomiting strongly suggestive of cyclical vomiting. She did not appear to grow during this period and a medical man said that the liver was enlarged. She did not walk until the age of 4 years and 3 months; since that time her general health has been fairly good, and from the age of 5 years she has grown steadily.

Physical examination: The patient is very bright and intelligent, her mental capacity being fully up to her age standard. Physically, she is rather undersized, and the development of the body is somewhat out of proportion to that of the limbs; height, 47 in.; spine, from occipital protuberance to tip of

coccyx, 20 in.; subcostal girth, 28 in. Skull normal; teeth show indentation on enamel of incisors, the margins of which are slightly irregular; other teeth are normal. The breath has a strong acetone odour. The liver edge is felt 3 in. below the costal margin, the edge being firm, but not irregular. The spleen is impalpable and not enlarged to percussion. Heart, lungs and nervous system are normal; systolic blood-pressure 105 mm. X-ray examination confirms the presence of an enlarged liver and shows nothing else abnormal. The blood-serum yields a negative Wassermann reaction (three tests).

The complete blood picture is as follows: Hæmoglobin, 80 per cent.; colour index, 0·9; red blood cells, 5,200,000 per cubic millimetre; no vacuolation but anisocytosis and poikilocytosis were marked. No normoblasts. White blood cells, 11,000 per cubic millimetre. Differential count: Polymorphonuclears, 54 per cent.; small lymphocytes, 26 per cent.; large lymphocytes, 14 per cent.; eosinophils, 4 per cent.; basophils, 1 per cent.; myelocytes, 1 per cent.

Urine: Specific gravity 1022, acid reaction, no albumin, no sugar. Acetone and diacetic acid both present. Centrifugalized deposit—a few epithelial cells, but no casts or crystals.

During the two and a half years she has been under observation she has remained fairly well, able to go to school and lead an ordinary life. The urine has been examined on many occasions, and both acetone and diacetic acid have invariably been present and sugar absent; also, as a rule, the breath has a strong acetone odour. Wassermann test made again (June, 1922), reaction negative; the blood picture showed no material change.

Several hepatic efficiency tests have been applied, all yielding negative results, thus:—

*Straus's Lævulose Test.*—The patient was given 60 grm. lævulose, and hourly specimens of urine were examined for six hours; in no specimen was sugar found present.

	Quantity	Specific gravity	Sugar	Acetone
(1) ... ..	1½ oz.	1022	Nil	+
(2) ... ..	3½ oz.	1018	"	+
(3) ... ..	8 oz.	1014	"	+
(4) ... ..	3½ oz.	1018	"	—
(5) ... ..	3 oz.	1020	"	—
(6) ... ..	1½ oz.	1028	"	++

It is to be noted that in the specimens obtained four and five hours after taking the lævulose, acetone was absent.

*Blood-lævulose Test.*—The blood sugar estimated by Benedict's method was found to be 0·109 per cent.: 25 grm. of lævulose were given by the mouth and the blood sugar estimated three quarters of an hour later. Result, 0·110 per cent. (Experimental error 0·005 per cent., certainly less than 0·01 per cent.)

*Widal Hæmoclasic Test.*—Total white cell count, 8,700 per cubic millimetre; one hour after 7 oz. of milk, 14,000 per cubic millimetre.

*Sulpho-conjugation Test* (Foster and Kahn's test).—Ratio of ethereal sulphates to inorganic sulphates in urine (benzidine method): Before thymol, 1 : 13; after ½ grm. of thymol, 1 : 4·4

The association of hepatic enlargement with the continued presence of acetone and diacetic acid in the urine, without glycosuria, is unusual. The history rather suggests a familial condition; the fact that both parents and the sister show no signs of specific disease and all, including the patient, yield negative Wassermann reactions, excludes syphilis. The familial form of

haemolytic jaundice is excluded by the absence of jaundice. As Sir Humphry Rolleston has pointed out ("Diseases of Liver," p. 445) congenital cystic disease of liver is known to occur in rare instances without the usual association of congenital cystic kidneys, and examples have been recorded by Borst, Dudgeon, W. Müller, and Batty Shaw. "Idiopathic" hepatic cirrhosis of children is also occasionally met with as a familial condition.

Although either of the two last mentioned disorders is a possible diagnosis, as far as I am aware, neither is usually associated with a persistence of acetone and diacetic acid in the urine. It has been suggested in this case that these substances may be of alimentary origin and their persistence possibly due to interference with the metabolism of amino-acids in a liver embarrassed by the formation of fibrous tissue. Against this view, however, is the fact that none of the hepatic efficiency tests indicate any defect of liver function. Increasing the carbohydrates in the child's diet does not lead to a disappearance of the acetone (although absent for a very brief period following the alimentary levulose test), and her mother is emphatic in the opinion that she is never as well when taking an increased amount of carbohydrate, stating that she becomes listless and irritable, and develops a poor appetite.

I desire to acknowledge my indebtedness to Dr. Clement Lovell for carrying out the various pathological investigations in the case, and to Sir Humphry Rolleston and Dr. Parkes Weber for their interest and helpful suggestions.

#### DISCUSSION.

DR. ERIC PRITCHARD (President) asked whether a similar case had been seen by any other Members. It seemed almost incredible that if the presence of acetone in the urine was of great importance—which was not his opinion—an acetonuria could go on indefinitely without producing a pathological change. Was that condition to be ascribed to any form of liver inefficiency, or was the converse true, that the liver became enlarged in an attempt to deal with this abnormal product of metabolism? The President considered it probable that the liver tried to oxidize the acetone and remove it from the blood.

Dr. F. PARKES WEBER said that when he saw this child in October, 1922, he was sceptical as to the urinary acidosis always being present, but on that occasion he obtained a specimen of urine which was free from albumin and sugar and gave positive Gerhardt's and Legal's reactions. Therefore he was no longer sceptical. The cyclic vomiting from which the child formerly suffered, and the idiosyncrasy towards butter from which she still suffered, indicated the presence of metabolic abnormality. He thought the persistent enlargement of the liver was connected with this metabolic disturbance. If the child's liver could be seen, he thought it would be found to show ordinary, but rather excessive, fatty infiltration, with, probably, an increase in size of the hepatic parenchyma. The liver was unable to deal efficiently with fat, and in such cases of inadequacy of parenchymatous tissue there was a tendency to compensatory hypertrophy or hyperplasia, to make up by increase in bulk for the inadequacy of the individual cell. In other words, the liver was enlarged, partly owing to fatty infiltration, and partly to an automatic effort on the part of the organism to compensate for qualitative defect of the parenchymatous unit (hepatic cell) by increase in the total parenchymatous (glandular) quantity.

Dr. R. HUTCHISON said he suspected that if the urine were examined oftener for acetone and aceto-acetic acid, it would be found that these substances were not uncommonly present. The type of child in whom they were likely to be present was the "livery" or "bilious" type of child who suffered from "hepatic inadequacy," who could not tolerate large quantities of milk or fats, and who frequently got cyclic vomiting or recurrent pyrexia or migraine. He always regarded these cases as having some

inefficiency of liver metabolism. He believed that the liver was the cause of the acidosis, and he agreed with Dr. Parkes Weber that if the liver in this case could be seen it would be found to be in a fatty condition. This was not necessarily the cause of the acidosis; it was due to the liver not dealing with the fats, but why that was so he could not say. With many people that inability lasted throughout life. He thought the present case was an extreme example of what was constantly being seen in less degree.

Dr. WORSTER-DROUGHT (in reply) said that the view expressed by the President had been considered and had much to commend it, especially as the hepatic efficiency tests—if of real value—indicated that the liver was functioning normally; consequently the enlargement might represent the effort on the part of the liver to deal with the acidosis. The urine had been examined on about 160 different occasions, and the only occasion on which acetone and diacetic acid were not found to be present was four and five hours after the ingestion of 60 grm. of levulose for the alimentary levulose test. Dr. Hutchison had mentioned that acetone was associated with biliousness, but in those cases the child was usually unwell at the time, whereas this child continued in fairly good health, and could lead her ordinary life.



## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

### Case of Pellagra.<sup>1</sup>

By R. HUTCHISON, M.D.

(ABSTRACT.)

THIS child presents a typical example of pellagra, showing an extreme degree of dermatitis, with the characteristic changes in the nervous system. The dermatitis is seen to be on exposed areas; it is also characteristic in that it recurs every spring, and disappears in winter. There is a great exaggeration of deep reflexes, and there is the curious mental change, the depression, which is well recognized in the disease. She has also slight optic neuritis.

As regards the cause, so far as investigation goes in regard to absence of vitamins or the giving of proteins of low biological value, neither of these hypotheses will explain this case; for from the age of 1 year this child has had an ordinary mixed diet, with plenty of milk, and sufficient fresh articles, and I have been unable to discover anything in the environment which could produce this condition.

### Case of Pellagra.

By DONALD PATERSON, M.B.

(ABSTRACT.)<sup>2</sup>

PATIENT, a girl, aged 11 years, admitted to Great Ormond Street Hospital, July, 1922, suffering from a rash and exhibiting clinically a gross nervous lesion. The rash had disappeared by October, and there has since been no recurrence. Mental state becoming more backward; patient has suffered from hallucinations.

#### DISCUSSION.

Dr. ERIC PRITCHARD (President) said that although he did not remember having seen a case of pellagra before, he had always taken an interest in the subject. Some time ago the Treasury sent an official to Egypt to make inquiries concerning certain economic reforms. This official had some knowledge of dietetics, and one of the subjects into which he inquired was the cost of maintaining inmates of the prisons and other institutions. He found the diet was what he considered liberal, and suggested its reduction. As a sequel, in about three years obscure cases of dermatitis arose among the inmates. Then the war came, and many Turkish prisoners were placed there, and an epidemic of pellagra occurred. Professor Wilson, the Professor of Physiology in Cairo University, instituted an inquiry, believing that the aetiology was associated with low protein value. He found that the biological value of the protein intake of the prisoners was about one-third of the physiological amount. When treated with a full biological allowance of protein the state of the cases rapidly improved, and he (the President) believed most of them were practically cured. He asked what was the usual course in children who were undergoing treatment.

Dr. SAMBON thanked the exhibitors for having given him the opportunity of seeing these patients. He had long been interested in pellagra, and never missed any occasion

<sup>1</sup> This case will be reported in full elsewhere.

<sup>2</sup> This paper will be published in full elsewhere.

that might yield him further information. Dr. Hutchison's case presented in a remarkable way all the characteristic symptoms of the disease. The symmetrical erythematous eruption affecting face, neck, back of hands, wrists, points of elbows and pudenda, with well-marked demarcation line, was typical, and its diagnostic significance was strongly confirmed by the associated gastric, nervous and mental symptoms, as also by the very clear history of the case. It was of peculiar interest to notice that the little girl had contracted the disease in the same place, near London, from which Dr. Box's case came in 1912. At the time he (Dr. Sambon) had visited the locality and found another unrecognized pellagrin child in the same family.

Some years ago it was thought there was no pellagra in this country. Two typical cases had been described, one by Dr. Howden in 1866, the other by Dr. Brown and Dr. Low in 1909, but Dr. Sandwith, who was an accepted authority on the subject and a redoubtable partisan of the maize theory of pellagra, had stated that the published accounts did not tally with the disease as he knew it; besides the patients had never been out of the British Isles, and maize was not their staple food, hence their disease could not be pellagra! It was a twice-told tale. It several other countries pellagra had been considered non-existent, only because it had not been previously recognized. When first discovered in Italy it was wrongly regarded as a new disease and, until quite recently, in American text-books, it was briefly dismissed for the reason that "it was not known to occur in the United States." Dr. Babcock and Dr. Watson, who, in 1907, first described cases from Columbia and Alabama, were ridiculed and abused, but five years later no less than 30,000 cases had been reported from thirty-three States! In his "*Traité de la Pellagre*," published in 1860, Billod mentioned two possible cases of pellagra seen in the extreme north-east of Scotland by Dr. W. A. F. Browne, Inspector-General of the Scottish Lunatic Asylum. The patients were two young idiots, and their symptoms seemed to tally with those of pellagra, but Browne disclaimed any knowledge of the disease.

Basing himself on this very meagre information and on the cases described by Howden and Brown and Low, he (Dr. Sambon) determined to make a rapid investigation. Being employed at the time, he had to ask for leave, and was grudgingly granted a week-end, from the Friday evening till the following Tuesday! Dr. Chalmers was also interested and went with him. They were away five days, and in that time they found several cases of the disease, and gathered sufficient information to prove that pellagra was and had long been endemic in the British Isles. On his return to London, Dr. Sambon found a letter from Sandwith awaiting him. It stated that at St. Thomas's Hospital, under the care of the Dean (Dr. Box), was a boy, who exhibited symptoms strikingly like those of pellagra, though he had never been farther than fifteen miles out of London, nor was known to have partaken of maize.<sup>1</sup> Straightway he (Dr. Sambon) went to St. Thomas's, and there saw a typical pellagrin. Dr. Box already had diagnosed his case correctly from photographs he had seen of American pellagrins.

On behalf of the Colonial Office and of the "Pellagra Investigation Committee" he had visited many endemic centres of the disease in different countries including Roumania, Hungary, Italy, France, Spain, the United States of America and the West Indies; he had found pellagra was the same all the world over, though environmental circumstances, and especially food, differed greatly from place to place. Everywhere he had found the disease occurring in the wealthy and well-nourished as in the poor and famished, in the robust as in the weakly, in breast-fed infants as in very old people. Having made a careful study of the epidemiology of pellagra, having noticed its peculiar distribution everywhere restricted to irregularly scattered, discontinuous stations characterized by rural conditions, the invariable immunity of truly urban centres, the striking seasonal incidence, two-fold in many places and always closely linked with seasonal, meteorological and other, no doubt biological factors, the characteristic periodicity of its symptom-triad, the irruption of recurrences invariably preceding new cases by at least a fortnight, the short incubation period, the strikingly different way the disease affected the sexes in different places according to the different occupations pursued in each, and so on, he must emphatically repudiate the prevailing dietetic conceptions of its ætiology.

<sup>1</sup> *Brit. Med. Journ.*, 1912, ii, p. 1095.

He would mention just one interesting observation he had been able to make in Italy some years ago. No doubt they could all recall the aspect of the Adriatic Gulf or *Mare Superum*. Venice, like all large towns, was immune from pellagra, but the disease prevailed extensively in the stream-raked rural districts on either side down to the sea-border. That these districts were true endemic centres was proved by the fact that the disease was rampant among the very young children, because, as he had shown and Chalmers and Lavinder had confirmed, in pellagra as in malaria, young children were the very index of the incidence of the disease. Among adults the disease here showed a decided preference for the female sex. Opposite Venice were the well-known islands of the Venetian Lagoon. Two of these, Murano and Burano, were of particular interest, both were little towns closely set with buildings and not a single patch of grass. Murano was famous, since the fourteenth century, for the manufacture of exquisite glass ornaments. All its inhabitants were employed in the making and packing of glass. They were by no means pictures of health, but pellagra was unknown amongst them. Burano also was famous on account of its wonderful lace. But lace was the work of women; the men were fisher-folk and during summer left their islet home to go fishing for weeks together along the mainland coast, sleeping at night up rivers either in their boats or in sheds on land where, at times, they were greatly tormented by mosquitoes and midges of many kinds. Pellagrins were numerous at Burano, but the little island-town was not an endemic centre of the disease as vouched by the fact that young children were never affected. Pellagra was practically unknown amongst the Burano women of all ages who never left the island except for rare day-trips to Venice and were constantly employed in lace-making. The disease was strictly limited to the male population including boys from the age of 10 or 12 years when, as a rule, they joined their parents or elder relatives in their fishing expeditions along the mainland coast. There were a few exceptions; thus he saw two pellagrin women, who, instead of making lace, used to go and labour in the fields of the mainland and some thirty men who worked as artizans at the Venice Arsenal. These men had never been out fishing and were free from pellagra. Professor Boni, who was physician to the Arsenal, stated that he had never seen pellagra in any of the Burano men who worked there. All brought with them the home-made polenta which was the staple food of the Lagoon islanders. How could one explain these remarkable facts by any of the prevailing "food"—"deficiency of food"—or "lack of vitamin"—theories?

He had not had the opportunity of doing any clinical, pathological or experimental work. Only once at Roman, in Roumania, together with Dr. Chalmers, he had been able to make the autopsy of a young woman who had died of acute pellagra. In smears from the brain and spinal cord they had found a minute organism not unlike a *Spiroschaudinna* in its endocarpuscular stage. Personally he regarded the organism as a protozoan and in this he was supported by Chalmers, Minchin, Leishman, Manson, Low and Laveran, but he did not feel justified in attaching any importance to an isolated finding. He urged that there should be a careful study of the aetiology of the disease. Pellagra was of more than academic interest to them. Since 1912, when he had first directed attention to the endemic prevalence of the disease in these islands, he had examined 128 indubitable cases. He had heard of many more and no doubt the unrecognized cases were numerous. Surely the two cases they had just seen made a very forcible appeal to their humanity and urged that so grave and fearful a disease, causing insanity when it did not kill outright, should receive urgent and well-directed attention.

Dr. F. PARKES WEBER asked what relationship the skin manifestations of pellagra bore to the other symptoms of the disease. Surely it was that the skin in this disease became sensitized? In England the sufferers from hydroa aestivale, when exposed to sun-rays, developed the hydroa on the parts exposed to the sun, as if their skins had been specially sensitized towards the solar rays. In a few of the cases there was hæmatoporphyrinuria, and apparently hæmatoporphyrin sensitized the skin to the rays of the sun.<sup>1</sup> If the cutaneous symptoms in pellagra were prevented from appearing by protecting the skin from the sun, did it make any difference to the course of the other symptoms of the disease?

<sup>1</sup> Cf. Mackey and Garrod, "On Congenital Porphyrinuria, associated with Hydroa Aestivale," *Quart. Journ. Med.*, Oxford, 1922, xv, p. 319.

Dr. H. STANNUS said that Dr. Paterson gave him the opportunity of seeing his case when the eruption was in evidence, and it certainly was a typical case of pellagra. With regard to the question of involvement of the nervous system, Dr. Sambon led one to believe there would be found changes of an infective character. As a matter of fact, the whole of the changes in the nervous system were of a toxic character; they were the same as those in sub-acute combined degeneration of the cord. The best description of the pathological changes in the nervous system in the disease was that by Dr. Kinnier Wilson, based largely upon the material the speaker brought home from Africa. Experiments made by injecting emulsions of cord and brain of pellagrins into monkeys were negative. This was done in six cases. He believed he was himself the first to describe the disease in Africa. The speaker had watched a number of cases for a year before recognizing the disease, and when he published them he had not completed his observations. They all occurred in a native prison in the middle of Africa, and the onset of the disease corresponded with the prisoners being given a change in diet, i.e., different from that with which they were supplied by their women folk, and the disease became very rife there ten years ago. After some difficulty the prisoners were put on to what was considered to be a correct diet, and after that, though other conditions remained the same, there were no fresh cases. He was a convert to the view that pellagra was in some way due to a deficiency, though he admitted the complexity of the problem. He believed that mere deficiency could not cause anything, but in the absence of some important element from the diet, whether it were some amino-acid or a vitamin, some abnormal products might be absorbed from the bowel which caused symptoms, such substance acting possibly on the endocrine glands. He regarded pellagra as a group of syndromes rather than as a disease. Dr. Hutchison's patient had a marked eruption on the extensor surface of the elbows; had that area been exposed? No doubt the rash was largely caused by the irritation produced by the sun's rays, but it must be remembered that the rash also occurred on parts of the skin which had not been so exposed. One of the earliest diagnostic features to which the speaker had first drawn attention was a little soddening of the epithelium at the junction of the inner and outer surfaces of the prepuce, a part which, in the native, was never exposed. Sometimes a similar condition was seen on the lips also, i.e., at the angles of the mouth. In the native this showed as a white area. In women the vulva was affected, in men the scrotum. Heat, moisture and pressure might be factors, and he wondered whether the child shown by Dr. Hutchison had been resting a good deal on its elbows. Dr. Sambon was a great enthusiast with his specific infective theory, but Goldberger in America had produced pellagra in men by feeding experiments, a fact which could not be lost sight of.

Dr. SAMBON (in reply to Dr. Stannus) said that no aetiological theory could be accepted unless it explained all the clinical, pathological and epidemiological features of the disease. Of the thousand and one dietetic theories so far advanced to account for the causation of pellagra not one took into consideration the characteristic periodicity of the disease, its peculiar topographic distribution, its two-fold seasonal incidence, its great prevalence among very young children, its remarkable diversities in sex incidence according to occupation, its short incubation period and the many other striking epidemiological features. Instead of studying true pellagra in the field, authors seemed to have set up imaginary pellagras in their laboratories. He thought the importance of the subject warranted a special discussion.

Dr. HUTCHISON replied that he was unable to answer Dr. Parkes Weber's question. With regard to Dr. Stannus's question, he would inquire whether the child's elbows had been exposed.

### Case of Chronic Splenomegaly of Uncertain Origin.

By F. PARKES WEBER, M.D.

THE patient, L. L., aged 8 years, is a well-developed dark-complexioned Polish Hebrew boy, who has recently been brought by his parents to England. He was admitted to hospital on February 27, 1923, on account of great

enlargement of the spleen, which probably had been present for a long while. There was apparently nothing else special in the past history. He had never been jaundiced and seemed to have enjoyed good health. Beyond the great enlargement of the spleen and slight anæmia nothing abnormal could be discovered by ordinary examination. There was no enlargement of superficial lymphatic glands; nor were there "Hutchinson's teeth" or other signs of congenital syphilis. The blood-serum gave a completely negative Wassermann reaction. There was nothing abnormal seen by ophthalmoscopic examination.

The patient's father and mother both look healthy; and so do the only brother, aged 10 years, and the only sister, aged 11 years, who are not jaundiced and have no enlargement of spleen, liver or superficial lymphatic glands. The mother has been pregnant only three times.

For the examination of the patient's blood I am indebted to Dr. G. Welsch. Blood count (February 27, 1923): Hæmoglobin, 60 per cent.; erythrocytes, 4,477,000 per cubic millimetre of blood; white cells, only 2,300 per cubic millimetre of blood (neutrophil polymorphs, 79 per cent.; lymphocytes, 12 per cent.; eosinophils, 1 per cent.; mast-cells, 1 per cent.; large mononuclears and transitionals, 7 per cent.). There was marked anisocytosis, but no definite poikilocytosis; no nucleated red cells seen. The resistance of the red cells towards hæmolysis (March 4, 1923) was estimated by adding drops of the whole blood to graduated hypotonic sodium chloride solutions. The hæmolysis was found to commence with a solution of 0.46 per cent. and to be complete with a solution of 0.4 per cent. There was therefore no marked excessive fragility of the red cells present. A second blood count (March 13, 1923) gave: Hæmoglobin, 65 per cent.; erythrocytes, 4,672,000 per cubic millimetre of blood; white cells, 3,500 per cubic millimetre of blood. The urine showed nothing abnormal: there was no excess of urobilin or urobilinogen. The fæces were normally formed and normally coloured.

With respect to this boy's case I thought at first one could say that he seemed "more splenomegalic than ill"—after Chauffard's saying regarding many patients with congenital hæmolytic icterus: "Plus icériques que malades." However, on March 17, 1923, he developed slight febrile follicular tonsillitis, which was followed on March 19 and 20 by considerable hæmatemesis and melæna. He is now (April, 1923) in his usual condition again, excepting for some increase in the anæmia as a result of the hæmorrhage. The blood count (Dr. G. Welsch, April 3) gives: Hæmoglobin, 49 per cent.; erythrocytes, 3,152,000 per cubic millimetre of blood; white cells, 2,800 (neutrophil polymorphs, 82 per cent.; lymphocytes, 16 per cent.; no eosinophils and no mast-cells; large mononuclears and transitionals, 1 per cent.). There is slight anisocytosis with slight polychromatophilia, but no poikilocytosis; no nucleated red cells have been found. The resistance of the blood to hæmolysis (April 5), estimated by the same method as previously, shows that hæmolysis commences with a solution of 0.48 per cent. and is complete with a solution of 0.38 per cent. When the *washed* erythrocytes are used instead of drops of the whole blood (April 10), it is found that hæmolysis commences with a solution of 0.36 per cent. and is complete with one of 0.2 per cent. The fragility of the red cells is therefore not excessive.

It should be mentioned that there has been some uncertainty as to whether the epigastric portion of the abdominal swelling is due to enlargement of the left lobe of the liver, or is part of the greatly enlarged spleen. It seems more probable that the liver cannot be felt, and that the whole swelling is a greatly enlarged spleen separated by a deep notch into an epigastric portion, which

extends considerably to the right of the middle line, and a lower portion, which reaches downwards almost to the symphysis pubis.

It is now recognized that congenital hæmolytic anæmia occasionally occurs as a variation of congenital hæmolytic icterus, in fact that it is the same disease only without the icterus. In 1909 I wrote<sup>1</sup> :—

"I have, however, satisfied myself that patients with congenital splenomegalic acholuric jaundice may sometimes (for a time at least) completely lose their icteric tinge. In other words, the jaundice may disappear and leave a condition of splenomegalic anæmia only. Moreover, in the same family one child may suffer from congenital splenomegalic acholuric jaundice, whilst another child may suffer from chronic splenomegalic anæmia without any manifest jaundice, though the blood-picture be the same in both children. Evidently, in such instances the splenomegalic anæmia is the same disease, whether it be accompanied by visible jaundice or not."

In the present case, however, owing to the absence of abnormal fragility of the erythrocytes, the complete absence of jaundice, and the absence of any excess of urobilin or urobilinogen in the urine, the case cannot be regarded as one of hæmolytic anæmia. It is more likely to be one of the Gaucher type of splenomegaly, but I have not ventured to confirm the diagnosis by splenic puncture and a search for the "Gaucher cells," with their small pycnotic nuclei and excess of clear cytoplasm. The question of the advisability of splenectomy of course arises. Personally, I am not in favour of the operation at present, because the boy's general condition is fairly good and the operation must necessarily be a dangerous one, and because the Gaucher type of splenomegaly is not a disease completely limited to the spleen, the Gaucher cells having been found also in the liver, lymphatic glands and bone marrow.

The patient must not be allowed to take part in rough games, such as football, which might easily lead to traumatic rupture of his enlarged spleen.

*Note.*—There is no transposition of viscera in the patient. Percussion of the area of hepatic dullness on the right side and a Röntgen-ray photograph make this clear.—F. P. W., May 3, 1923.

### Congenital Hæmolytic Jaundice.

By F. PARKES WEBER, M.D.

THE patient, W. P., aged 12 years, is a well-built and healthy-looking English boy, but with a slightly icteric tinge of the sclerotics. The spleen can be felt enlarged, reaching just below the costal margin. The liver is apparently not enlarged. The fæces are brown and normal in appearance. The urine is of specific gravity 1020; clear; of orange colour; free from albumin, sugar and bilirubin, and giving positive reactions for excess of urobilin and urobilinogen. For the careful blood examination I have to thank my house-physician, Dr. G. Welsch: Hæmoglobin, 60 per cent.; erythrocytes, 3,952,000 per cubic millimetre of blood; white cells, 6,000 (neutrophil polymorphs, 61 per cent.; lymphocytes, 33 per cent.; transitionals and large mononuclears, 2 per cent.; eosinophils, 4 per cent.; no mast cells). There is marked anisocytosis, slight poikilocytosis, but no polychromatophilia; no nucleated red cells have been detected. The resistance of the erythrocytes to hæmolysis, as estimated by adding drops of the whole blood to graduated hypotonic sodium

<sup>1</sup> F. Parkes Weber, "Acquired Chronic Acholuric Jaundice," *Amer. Journ. Med. Sci.*, Philad., 1909, vol. cxxxviii, p. 24, footnote 3.



chloride solutions, is found to be definitely below the normal, hæmolysis commencing with a solution of 0.58 per cent., and being complete with one of 0.46 per cent. The blood serum gives a completely negative Wassermann reaction, and in all other respects (including ophthalmoscopic examination) the boy appears to be normal. There has never been any icteric pruritus or xanthoma. He is one of a family of three children; the other two children and the mother and father are apparently not subjects of hæmolytic icterus. When 6 months of age the patient was supposed to have "splenic anæmia" and purpura, but the purpura soon disappeared. Since then, however, he has been subject to attacks of vomiting, accompanied by obvious yellowness of his skin and sclerotics, and by a feeling of great feebleness. These attacks have occurred about twice yearly and have lasted on the average about two weeks, but he has likewise had similar lesser attacks of short duration.

The case is a typical one of hæmolytic icterus of moderate degree, with relatively little enlargement of the spleen and not much anæmia. I am inclined to think that slight cases of hæmolytic icterus (or hæmolytic anæmia, without definite icterus) are more frequent than is commonly supposed and that they are not rarely overlooked, even by skilled physicians. This opinion is due partly to my having read a recent paper on the subject,<sup>1</sup> and partly to my colleague, Dr. E. Schwarz, having given me the opportunity of examining with him a girl with typical hæmolytic icterus and also her maternal uncle. We found that the sclerotics of the latter had a very slight icteric tinge, that his spleen was slightly enlarged, and that his urine showed a definite excess of urobilin and urobilinogen. He evidently, like his niece, was a subject of hæmolytic icterus, but this had not been recognized by the doctors who had previously been consulted by him for certain attacks that were regarded as attacks of cholelithiasis or cholecystitis. It was easy for us to make the diagnosis, as we knew that his niece was a subject of typical hæmolytic icterus. In a family group of cases of hæmolytic icterus which I described with Dr. G. Dörner in 1910, the condition in two of the cases (especially at a time when there was hardly any jaundice present) might have been easily overlooked.<sup>2</sup>

#### DISCUSSION.

Dr. R. HUTCHISON advised splenectomy in the first of these cases. The two tumours he considered were the left lobe of the liver, and the spleen. He did not know why Dr. Weber diagnosed the Gaucher type; this was very rare, and it was better not to diagnose a rare condition unless one felt very sure about it, and here a clear family history was absent. But even if it were the Gaucher type, that constituted an additional reason for removal of the spleen, because it was practically an endothelioma of the spleen. He had had one case of the Gaucher type. Splenectomy was done, and the child did well. The first of these cases he regarded as splenic anæmia of the adult type; these cases proceeded to Banti's disease, and died at puberty of profuse gastro-intestinal hæmorrhage. Splenectomy in the second case would not get rid of the fragility of red cells, but it would greatly lessen their destruction.

Dr. H. STANNUS said large numbers of cases of splenomegaly occurred in the tropics; he had seen villages in which 60 per cent. of the children were so afflicted; it was not malaria nor was it kala-azar. He believed the patient in the first case shown had a large left lobe of the liver and an enlarged spleen. He knew practically nothing

<sup>1</sup> A. M. Gänsslen, "Ueber hämolytischen Icterus," *Deut. Arch. f. klin. Med.*, Leipzig, 1922, cxl, p. 210.

<sup>2</sup> F. Parkes Weber and G. Dörner, "Four Cases of Congenital Acholuric Jaundice (so-called Hæmolytic Jaundice) in one Family," *Lancet*, 1910, i, p. 227.



about the aetiology of these cases in the tropics (the Wassermann reaction was negative) nor anything of what happened to them later, as he only saw them during his travels. Their aetiology was probably similar to that of the case shown. In the African cases the relative size of the right and left lobes of the liver and of the spleen was very variable.

### Argyll-Robertson Pupils with Mydriasis.

By F. PARKES WEBER, M.D.

THE patient, A. S., is a fairly well-developed boy, aged 13 years, who has very large pupils which do not react to light. The right pupil is not quite as large as the left and is not perfectly circular. Both pupils react to accommodation, but rather slowly. By ophthalmoscopic examination both fundi appear normal, and in other respects nothing else can be found abnormal in the boy, excepting in regard to his tendon-reflexes. A knee-jerk can be obtained on the right side (at all events by the "reinforcement" method), but not on the left side. The Achilles tendon-jerk cannot be obtained on either side. The plantar reflex is of the normal flexor type on both sides. The blood serum (March, 1923) gives a completely negative Wassermann reaction.

In the absence of examination of the cerebro-spinal fluid the possibility of the condition being the result of slight nervous lesions due to congenital syphilis (neuro-syphilis) cannot be absolutely excluded. Dr. C. Markus, however, to whom I am indebted for the present case, described in 1906<sup>1</sup> somewhat similar cases of partial iridoplegia in which no absolute evidence of syphilis was obtained. In his cases, then described, the condition was present in only one eye, and over-action of the sphincter iridis on accommodation was a feature. His first case was that of a boy (F. S.), aged 12 years, whose right pupil was very large and did not react to light, though it contracted (somewhat sluggishly) to accommodation. Nothing else abnormal was found in him excepting in regard to his tendon reflexes. His knee-jerks and his Achilles tendon-jerks were absent on both sides. The plantar reflexes were of the normal flexor type. In a man with pulmonary tuberculosis, whom I saw (1910), there was a unilateral Argyll-Robertson pupil of medium size, on the right side, and the right knee-jerk was less than the left one. I did not exclude syphilis as the possible cause.

The family history of the present case (A. S.) is that the father died at 40 years of "heart-stroke." The mother, living, aged 50 years, has had four children and four miscarriages. Of the four children, one, besides the present patient, is living, namely, a daughter, aged 20 years, said to be healthy. One, a boy, died at 18 months of "convulsions." The other, a boy, died at 13 years of tuberculous meningitis.

P.S.—The cerebro-spinal fluid obtained by lumbar puncture, on May 10, 1923, was perfectly normal and gave a completely negative Wassermann reaction.

C. Markus, "Notes on a Peculiar Pupil Phenomenon in Cases of Partial Iridoplegia," *Trans. Ophth. Soc., Lond.*, 1906, xxvi, p. 50.

### **Case of Heart Disease.**

Shown by G. M. KENDALL, M.B.

(For Dr. A. C. D. FIRTH.)

PATIENT, a boy, aged 6 years, who was apparently healthy at birth and up to 3 months of age. When 3 months old he had a fit during which he went blue. At 4 months of age he had bronchitis and has had a cough ever since. When 6 months old he was examined by a doctor who said the heart was affected. He attended a Welfare Centre regularly from then until 1½ years of age and his mother was told several times that he had "heart trouble." At 1 year and 3 months of age he had diphtheria and the mother was again told by the doctor at the Fever Hospital that his heart was diseased. Since he was 3 years old he has had attacks of blueness and difficult breathing. He often has sore throats and sometimes complains of pains in the legs.

Present condition: He now shows moderate cyanosis and well marked clubbing of the fingers and toes. There is frequent cough and often epistaxis. The lungs show signs of bronchitis. The pulse is regular and varies between 100 and 120. The heart is greatly enlarged to the left and slightly downwards. There is no apparent enlargement to the right and the upper limit of cardiac dullness is in the fourth space. The impulse is visible and forcible. There is a diffuse systolic thrill and a loud systolic murmur all over the precordium. The murmur is heard best over the apex but is not appreciably conducted into the axilla. At the apex there is also a short rumbling mid-diastolic murmur. The liver is not enlarged. There is no oedema.

X-ray examination: The skiagram of the chest shows a heart which is much enlarged to the left, and gives evidence of pleuro-pericardial adhesions in the right cardio-phrenic angle: the right side of the diaphragm is fixed.

Diagnosis: Congenital heart disease or rheumatic carditis in addition.

Dr. MURIEL RADFORD showed a Case for Diagnosis (Prominence of Right Lower Anterior Thoracic Wall in a Female Infant aged 9½ months). Case discussed by the President (Dr. ERIC PRITCHARD).

### **Two Cases of Osteogenesis Imperfecta.**

By BERNARD MYERS, C.M.G., M.D.

THE two patients who exhibit this disease are brothers, aged 5 and 2½ years respectively. They have recently been treated at the Royal Waterloo Hospital. The elder boy, R., would appear to be a case of osteogenesis imperfecta congenita, and although the case of the younger one, J., fits in with the diagnosis of osteogenesis imperfecta tarda to a certain extent, it is not unlikely that both are really cases of the congenital variety.

Family history: The father states that for three or four generations at least there have been bone deformities in various members of the family. A sister of the father is stated to have a fiddle-shaped chest and to be very small. Another sister has curving of the bones of the forearm, and her son again is

suffering from achondroplasia. The mother of these two patients had first of all a miscarriage, then seven children all living and varying in age from 20 years to 7 months: all of them normal except the two patients.

*Case I.*—R. G. H., aged 5 years has not yet walked alone. The mother states that she was very terrified during the air-raids. The birth seems to have been quite normal. The child was breast-fed until a year old, and the mother states that she had plenty of milk. After that the diet consisted of milk, Quaker oats, and whatever was procurable. Formerly the child suffered from head-sweating and was restless. The bowels are opened daily and the appetite is good. The parents are quite certain that R. was born with deformities, very similar to the condition which is seen at present. He has had measles.

When examined on admission to hospital it was noted that he was deformed and small for his age, the measurements being:—

		Cm.	Cm.
Height	...	73.0	105.6
Sitting height	...	48.0	59.5
Length of legs	...	25.0	46.0
Head: Circumference	...	48.5	50.0
Longitudinally	...	16.9	17.2
Laterally	...	13.2	13.4
Chest	...	45.5	52.3
Abdomen	...	50.5	50.9
Intercristal diameter	...	14.0	17.5

The measurements in the right hand column represent normal measurements found for males of similar age at the children's clinic.<sup>1</sup> The shortness of the legs was accounted for by fractures and deformity, and the pressure must have accounted, to some extent, for the decreased intercrystal diameter.

The bones of the arms and legs are noted to be distinctly deformed. The boy is backward for his age as regards his mentality and his talk is rather unintelligible, although he has improved in this respect since admission to hospital. The teeth erupted late and are at present small and decayed, with many absent. The child is pale, and sits with his legs crossed. When helped to stand, his feet are placed together, the knees are bent and there is some bowing of the legs. On the left tibia there is a swelling about its middle suggestive of what has been called annular rickets. The femora curve forwards and outwards and the tibiae have also a similar curve. The abdomen is distinctly enlarged and the liver and spleen were palpable upon admission to hospital, but they are not felt now. The chest protrudes anteriorly and is fiddle-shaped. Small protuberances are felt, similar to the rachitic rosary, there is a Harrison's sulcus, and also a costal flare; the costal angle is of greater width than normal. Kyphosis is present and particularly marked about the centre of the dorsal region. There is however no angular curvature. The child sits in a huddled position with back bent, head and shoulders forwards and legs crossed. He seems to be nervous of falling over.

Each arm shows a remarkable deformity in the upper part of the humerus where there is an inward and forward curve with a concavity posteriorly. The forearms are outwardly curved and thickened at the lower epiphyses. The head is perhaps a little broader than normal and gives the impression of having a small ridge above each ear. The calvaria membranacea, which is generally believed to be typical of this disease, is not present in this case. The sclerotics are bluer than normal. The mouth is generally open. The tongue is clean

<sup>1</sup> Bernard Myers, "Practical Handbook on Diseases of Children," H. K. Lewis.

and nothing abnormal was noted in the throat. Glands are palpable in the anterior and posterior triangles of the neck. So far no satisfactory skiagrams of the skull have been obtained as the boy had to be sent to a Metropolitan Asylums Board hospital, suddenly, as a diphtheria carrier. The X-rays show the long bones of the lower extremities to be more porous than normal and to contain less lime salts. The cortex is thinner than normal and the medullary spaces appear to be enlarged. At the lower end of each femur there are irregular lines at the junction of the diaphyses and epiphyses with apparently excess of cartilage and not much bone formation in the epiphyses. In fact the lower epiphyses are rather suggestive of rickets. Fractures are seen towards the middle and upper part of each femur; some of the fractures show a good deal of callus formation, but this would appear to be rather deficient in lime salts and the bone formed is evidently of poor quality. Each tibia also shows a fracture about the centre of the shaft. On the right side the fibula is also similarly affected and there is apparently not very much callus formation. The skiagram of the arm shows a want of calcium salts, a thin cortex and comparatively large medullary cavities. Many fractures are visible, some showing much more callus than others. The end of the radius shows a cup-shaped formation with excess of epiphyseal cartilage and very little ossification. Nothing abnormal was noticed in the heart or lungs. The child looks a little anæmic, but the blood count has not yet been made. There is no jaundice, cyanosis or dyspnoea. The muscles are generally flabby; no alteration in the knee-jerks was noticed. The Wassermann reaction was negative. The temperature showed slight irregularity during the period the child was in hospital, varying from  $97^{\circ}$  to  $100^{\circ}$  F., the daily variation extending from  $1^{\circ}$  to  $2^{\circ}$ .

Dr. Archer found nothing of importance in the calcium and glucose content of the blood nor in the urinary and faecal constituents. There are at least twenty fractures seen by X-rays in the upper and lower extremities and in addition there is evidence of a fracture of the right clavicle and also numerous fractures in the ribs. As far as can be ascertained all the fractures are intraperiosteal.

Diagnosis: Achondroplasia, cretinism, mongolism, hereditary syphilis and scurvy are negatived by the clinical signs and X-rays appearances, and osteomalacia by the history. Notwithstanding that the head is not typical this case appears to be quite clearly one of osteogenesis imperfecta congenita, with the addition of some of the signs of rickets in the epiphyses of some of the long bones. The changes in the ribs might conceivably be due to one or the other disease.

Case II.—J. H., aged  $2\frac{1}{2}$  years, shows the same condition as R., but to a less degree. There was nothing special about the birth of this child. He was breast-fed for twelve months and then given glaxo. He walked until a few months ago, when he had diarrhoea, vomiting, and cough, for which he was treated at Great Ormond Street Hospital. This child also shows several fractures in the long bones and ribs. The chest is fiddle-shaped, and is drawn in at the sides on inspiration. The head is square-shaped, like that of his brother. The tonsils are enlarged. The child is mentally not quite up to the normal standard for his age, and is backward in speech. Nothing abnormal was noted in any of the internal organs. The X-rays show that the cortex of the long bones is a little narrower than normal, and is irregular, the medullary cavity being again comparatively a little larger. The lower epiphyses of the femur show more bone formation than in the case of his elder brother. There are fractures about the centre of each femur with a good deal of callus formation, but there is some rarefaction at the site of the fractures. The upper arms

## 72 Myers: *Osteogenesis Imperfecta*; *Absence of both Thumbs*

show a porosity of the bones with fractures, in which some show more callus formation than others. The lower epiphyses of the radius are cup-shaped, and show excess of cartilage and not much bone formation. Fractures are noted in the ribs.

The child, although suggestive of being a case of osteogenesis imperfecta tarda with a certain amount of rickets, might have been a congenital case, as the parents may have missed less marked changes which may have previously been present.

### DISCUSSION.

Dr. ERIC PRITCHARD (President) asked whether these cases presented the marked characteristics of true osteogenesis imperfecta, the parietal ridge, which John Thomson regarded as the typical deformity? Also was there any thickening of the shaft of the bone? He himself had in his ward a patient with typical osteogenesis imperfecta, but without the parietal ridge. The incidence of the condition seemed to be selective, as all the bones were not affected; the tibia and fibula on one side seemed to be normal. On the other side there was marked thickening of the periosteum, there were fractures, and the ends of the bones were more chaotically osteoporotic than would be expected in infants. His case was sent in as an extreme instance of scurvy rickets, owing to the general tenderness, and only on X-ray examination were the multiple fractures discovered.

Dr. R. HUTCHISON said there was a great element of rickets in these cases, though he did not know whether that would explain the fractures. He did not regard the cases now shown as typical of osteogenesis imperfecta; the shape of the head and other features of the cases did not support that view. But these imperfections of bone were very difficult to classify, and would remain so until more was known about the pathology.

Dr. MYERS (in reply) said that Sir Arthur Keith had suggested to him that he should ask Mr. Knaggs's opinion of the two cases. After examining them Mr. Knaggs said he had no doubt that both children were suffering from osteogenesis imperfecta, even in the absence of the typical skull condition, but all the same the general appearance of the skull was rather suggestive. He considered there was a certain amount of rickets present also. Family cases of osteogenesis imperfecta, rickets and achondroplasia had been described, and it was difficult to understand why one member of a family should develop bone peculiarities in one direction and perhaps brothers, sisters or cousins develop abnormalities in another direction.

## Absence of Both Thumbs, with other Deformities of the Upper Extremities in an Infant.

By BERNARD MYERS, C.M.G., M.D.

PATIENT, a female, aged 4 weeks, has no thumb on the right hand, and most of the radius is absent. The X-rays show that four metacarpals are present, the upper portion of the radius is seen to be synostosed to the ulna, the remainder of the radius not being seen. On the left hand there are three fingers only, the thumb and index finger being absent, together with their metacarpals, and the whole of the left radius. The right humerus appears to be of normal size, but the left is diminutive. There are no other bony or soft tissue abnormalities, and the infant's health is good. Mr. Fairbank is treating the child surgically. No other relatives have bony deformities.

## Section for the Study of Disease in Children.

President—Dr. ERIC PRITCHARD.

### DISCUSSION ON BIRTH INJURIES.

#### (A) CASES.

#### **Case of Birth Injury to Brachial Plexus; all Cords of Plexus originally involved; Recovery of Function in Outer and Posterior Cords; Paresis now of Infraclavicular or Klumpke Type.**

By C. WORSTER-DROUGHT, M.D.

M. C., AGED 2 years, was first seen in July, 1921, when 3 months old, with a completely paralysed right arm; the condition had been present since birth. She was a full-term first child, with breech presentation, and she is stated to have been born with the arms extended at the side of the head. No instruments were used and no anaesthetic was given for delivery.

On examination there was found to be a flaccid paralysis of the entire right arm including the deltoid and pectoralis major muscles; as far as could be ascertained in a child so young, the scapular muscles were not affected. Electrical reactions: To faradism no response was obtained in any of the paralysed muscles; to galvanism (the child being under general anaesthesia) all muscles showed some reaction excepting the intrinsic muscles of the hand. In the pectoralis major, deltoid, biceps and triceps, the response was fairly good with kathodal closing contraction greater than anodal closing contraction; in the extensor and flexors of the wrist and fingers the response was poor, even with a strong current, anodal closing contraction was greater than kathodal closing contraction. The child did not appear to feel pin-pricks below the elbow and on the inner side of the upper arm, but admittedly sensation was difficult to test. X-ray examination showed nothing abnormal.

It was clear that all cords had been stretched and damaged by upward traction of the right arm, and as one would expect, the inner cord had suffered most. The limb was suitably splinted at a right angle at the shoulder and the forearm supinated and flexed. Treatment by massage and galvanic baths was then instituted.

Since this time the state of the limb has slowly progressed towards recovery. After two months (September, 1921) extension of the first and second fingers was noted, this being the first movement to appear. Before the end of 1921, voluntary movement appeared in pectoralis major, deltoid, biceps, and extensors of the arm. This was followed by extension of the forearm and



## 74 Worster-Drought: *Birth Injury*; Perkins: *Erb's Paralysis*

wrist (February, 1922), and by March, 1922, all the muscles above the level of the elbow reacted to faradism.

The muscles of the upper arm continued to develop, and by May, 1922, the extensors of wrist, fingers and thumb were practically in their normal state and all reacted to faradism. The flexors of wrist and fingers and intrinsic muscles of hand remained as before and it was not until February, 1923, that slight and occasional flexion was noticed in the first and second fingers and a reaction with strong faradism could be obtained in the flexors of wrist and fingers.

At the present time the only muscles that remain paralysed are the intrinsic muscles of the hand, but the flexors of the wrist and fingers are still weak and there is no hand-grip. The attitude of the arm is that of rotation outwards and hyperpronation. The flexors of wrist and fingers react to strong faradism and to galvanism, anodal closing contraction being greater than kathodal closing contraction; the abductor pollicis also reacts to faradism but no reaction can be obtained in the interossei or hypothenar muscles. Sensation appears impaired on the ulnar side of the hand and the inner aspect of the forearm.

It is thus seen that all roots have recovered function with the exception of those derived from the eighth cervical (flexors of wrist and fingers) and first dorsal nerves (intrinsic muscles of hand).

Though the limb has recovered so far, the intrinsic muscles of the hand supplied from the inner cord, still fail to react to any form of electric stimulation, and it is now a question whether exploration is advisable for the treatment of the inner cord. I should be interested to hear opinions on this point.

Paralysis of the lower arm (Klumpke type of birth palsy) is somewhat rare, over 80 per cent. of cases of birth injury to the brachial plexus being of the upper arm, or Erb-Duchenne type. Even when the whole plexus is at first affected and some recovery ensues, the residual palsy is often of the supraclavicular (Erb-Duchenne) type. In ninety-four cases of birth injury to the brachial plexus, Stransky found only twelve of the Klumpke type, and Thomas was unable to collect more than sixteen examples. According to Sherren, this by no means represents its true rarity, for many of the common upper-arm types of case are not recorded.

Spontaneous recovery has taken place in about 70 per cent. of cases of brachial birth plexus injuries (Sherren), but recovery will rarely be complete if no improvement has taken place up to the age of three months.

### Case of Erb's Paralysis.

By G. PERKINS, M.Ch., F.R.C.S.

THIS boy was first seen in February, 1921, when five years of age. No history of previous treatment could be obtained. He exhibited the muscular contractures commonly associated with a neglected case of Erb's palsy. The shoulder was adducted and internally rotated, the forearm extended and pronated. All the muscles of the limb responded to faradism, but there was no voluntary power of abduction or external rotation at the shoulder, and no power of supination of the forearm. The joints were normal. Mr. W. Rowley Bristow operated in May, 1921, and divided the tendon of the subscapularis muscle; in this way full passive range at the shoulder was obtained. The



arm was then put up in a plaster of Paris splint with the shoulder abducted 90 degrees and externally rotated, the elbow flexed, and the forearm in full supination. This posture was maintained for one year, with the addition of physiotherapy. Now, the boy has a fairly useful arm. At the shoulder, full passive, and half active abduction are possible; but there is scarcely any active external rotation. The elbow can be fully flexed, and at the forearm full passive and half active supination are possible.

### **Case of Spastic Hemiplegia.**

By G. PERKINS, M.Ch., F.R.C.S.

THE mother of this child states that she noticed dragging of the left foot as soon as the child began to walk. There is no history of difficult labour, or even of instruments being used at birth. When seen in January, 1921, the girl was 8 years old; she had a condition of spastic hemiplegia on the left side; the forearm was held in spasm pronated, and the foot plantiflexed. In January, 1921, Mr. W. Rowley Bristow performed a Stoffel operation on the right internal popliteal nerve, dividing the nerve-supply of the gastrocnemius, and half that of the soleus muscle. In February, 1921, a Stoffel operation on the median nerve was done, and the nerve-supply of the pronator radii teres and the flexor carpi radialis divided. The child has since undergone physiotherapeutic treatment. Now, she walks with a normal gait, and is able to dorsiflex the ankle to 90 degrees. There is three-quarters active supination in the forearm, and three-quarters active dorsiflexion at the wrist. The elbow moves from 90 degrees to full flexion. The function of the arm is poor owing to marked inco-ordination. Athetosis is present, and the child uses the left hand for all ordinary purposes.

### *(B) DISCUSSION.*

Dr. B. MYERS, C.M.G.

said it was a source of much satisfaction to know what surgery could do to improve deformities resulting from birth injuries. He had hoped to bring before the Section a boy, aged 6 years, who suffered from spastic paraplegia, the result of a birth injury. The mentality being only a little below the normal (an important matter), it was decided to operate. The boy had the typical gait. Tenotomy was done in the adductor longus and the tendo Achillis on each side, thus allowing the legs to be separated and the heels to come down. Next, Stoffel's operation was performed on the calf muscles, about 50 per cent. of the nerves concerned being cut. The final result had been so successful that the boy, who formerly could only attempt to walk with great difficulty, was now able to walk and run, although he was just a little stiff in starting off. Mr. Rocyn Jones could be well satisfied with the excellent results obtained.

Mr. Perkins remarked that there was no birth injury in his case. He (Dr. Myers) would shortly be publishing a case with Dr. Kirkwood, which demonstrated the difficulties sometimes present in determining the presence of tiny hæmorrhages.

The patient, a boy, was born of a primipara, aged 25, and was moderately cyanosed at birth. The labour lasted twelve hours, no instruments were used, and the mother was only given a few whiffs of chloroform. He saw the infant on the second day, when it had a slight right spastic hemiplegia. While being observed it had an attack of inspiratory apnœa which lasted about twenty seconds, and then normal breathing was resumed. These attacks occurred from time to time during the day, and at night the child died. Recently, in the *Journal of Physiology*, Dr. Thomas Lumsden had published a most interesting article describing his experiments on the respiratory centre of cats, and so closely did this case resemble some of Dr. Lumsden's results, that a necropsy having been allowed, he (Dr. Lumsden) was asked to investigate the brain. The diagnosis was hæmorrhage of the cortex and a lesion in the respiratory centre, of an unknown nature. On the surface of the brain of this child were found fine punctiform hæmorrhages. Macroscopic inspection of the brain-stem revealed nothing, but when it was examined in sections there were found in the pneumotaxic centre which Dr. Lumsden had described several hæmorrhages, the effect of which had been similar to that which obtained in his experiments on cats. Had it not been for Dr. Thomas Lumsden's work there would have been no suggestion to investigate this case microscopically. He (the speaker) had been wondering whether such hæmorrhages were not more frequent than they had been supposed to be; doubtless often a microscopic examination would be needed to render them visible. Six weeks ago an article by Cruickshank appeared in the *Lancet*, in which he expressed his opinion that birth hæmorrhages were not infrequent. He would like to hear whether other members had seen this inspiratory type of apnœa, and whether the brain-stem had been examined for tiny hæmorrhages.

In answer to the President, he said the pneumotaxic centre was in the pons, just above the apneustic centre. Immediately below the latter was the "gaspig centre." The region to which he was referring was just above the junction of the pons with the medulla.

#### Dr. F. C. SHRUBSALL

said he was able to give some figures from the data collected in the course of medical examinations in the special schools of London and from examinations under the Mental Deficiency Act.

In the schools for the physically defective, out of 4,500 cripple children investigated, the paralysis had been found to date from birth in 169 cases, and of these 157 were cases of spastic paralysis. The proportions were nine as of hemiplegia to seven of diplegia. Of course it must not be assumed that these cases were due to birth injuries, although noted from birth. In the schools for the mentally defective there were about 2 per cent. of cases of cerebral paralysis noted from birth. In the cases examined under the Mental Deficiency Act there was a more accurate history since there were special officers who interviewed all parents and traced back the early history as far as was possible. Adequate records were available in 146 cases equally divided between the sexes, the distribution of the paralysis and the birth history being as follows :—

				Difficult or protracted labour	Asphyxia	Normal labour
Males	{	Hemiplegia	...	8	3	33
		Diplegia	...	7	—	25
Females	{	Hemiplegia	...	10	4	22
		Diplegia	...	8	4	30

In two cases of diplegia in female children and in one case of hemiplegia in a male child the asphyxia was the only factor; in the others the labour had been protracted.

In 233 cases of spastic paralysis associated with mental impairment in which the information to hand was somewhat less detailed it appeared that in fifty-one there had been protracted or difficult labour, in thirteen difficult labour and asphyxia and in five asphyxia alone; while in 177 the confinement was uneventful.

If the mentally defective, taken as a whole, were considered it was found in cases other than those with paralysis that there was protracted pressure in 9 per cent. instrumental delivery—without special reference to prolonged parturition in 5·8 per cent. and a history of asphyxia in 2·8 per cent. There were of course insufficient grounds for asserting that these conditions caused the mental defect, in the absence of further evidence, but it was in line with the views of some Italian psychiatrists who held that even mere backwardness was often due to minute meningeal hæmorrhages caused at birth. On the matter of the intellectual status associated with different types of paralysis to which reference had been made in the discussion, the figures obtained by Dr. Carleton Williams in the schools for the physically defective showed the following proportions per cent.:—

Mental status, morbid condition		Average and over average		Below average		Bordering on mental deficiency
Infantile paralysis	...	66·9	...	25·6	...	7·5
Hemiplegia	...	41·2	...	30·9	...	27·9
Diplegia	...	15·1	...	24·2	...	60·6

the relation between intellectual failure and physical damage being very marked.

#### MR. C. MAX PAGE, D.S.O.

said he had not a wide experience of the surgery of the kind of cases which were under discussion, but he could say that exploration of the brachial plexus in these patients was practically valueless. In general, operations on the upper extremities for such injuries were of use only in so far as they remedied position, as in cases of dislocation of the shoulder, enabling coarse movements of the shoulder, elbow and radio-ulnar joints to be carried out. He had never seen a case in which surgery had materially improved the fine movements of the hands which were so all-important. Mr. Perkins stated that surgery might be successful in regard to contractures of the forearm, but that the functional results were indifferent. In the case of the lower extremities, however, the movements were coarse and largely automatic, and Stoffel's operation was very valuable.

#### DR. E. BELLINGHAM SMITH

said that in regard to these cases diagnosis was important. It seemed to have been taken for granted that the cases of hemiplegia and diplegia which had been seen in schools were instances of birth injury. He had seen many of these cases, and had had some difficulty in being certain that the condition was

due to injury at birth, especially when the child was some years old when he first saw it. The same condition of spastic hemiplegia and spastic diplegia could be produced by polio-encephalitis in the very early years of life, certainly after the age of 6 months. He thought that once the suggestion was made to the parents that the condition originated at birth, they would remember that the child had been a little blue when born, as so many children were, or that instruments had been used, or that the birth had been a prolonged one, and hence the case came to be attributed to palsy due to hæmorrhage at birth, or to a prolonged period of apnœa, in which either small hæmorrhages occurred, as had been described, or a definite change occurred in the brain substance, which later led to sclerosis. He thought that that must account, in Dr. Shrubbsall's list, for the small number of instances of damage at birth or instrumental delivery. Probably many of the cases referred to by Dr. Shrubbsall were examples of polio-encephalitis. He would like to hear the experience of other members on this point. In the case of the boy shown by Mr. Perkins it was not noticed until the boy began to walk, and he (the speaker) would have thought it was not necessarily birth injury, but a case of polio-encephalitis.

#### Dr. WORSTER-DROUGHT

(in reply) said that he agreed with Dr. Bellingham Smith that cases of cerebral birth injury were not always easy to diagnose. It was necessary to distinguish them not only from polio-encephalitis, but from cortical thrombosis following exanthemata such as measles. Also, there was a congenital syphilitic form of cerebral sclerosis which simulated cerebral diplegia of traumatic origin very closely.

With regard to operations on the inner cord of the brachial plexus, in war wounds where there had been a gunshot injury of the inner cord, he had seen in many instances much good result from freeing this cord from compressing and adherent scar-tissue. In this case, he thought, the sheath of the inner cord had been torn, causing hæmorrhage, and that the cord was now involved in scar tissue, rather than severed.

#### Mr. G. PERKINS

(in reply) said a point that had been raised was the element of intelligence as a determinant of surgical interference. He did not think that that should be taken into account, as even an idiot could walk; and if by means of a Stoffel operation in a case of spastic diplegia the patient was enabled to walk, there was at least the satisfaction of having been able to do something to remedy the evil. The orthopædic surgeon, as a rule, did not specialize in pathology, and he treated these cases of spastic diplegia as of one class of infantile spastic palsies. The operator's idea was that there was an upper motor neuron lesion, for example, at the ankle-joint, where the muscles played over it. The muscles in the front were weak, those at the back were strong; normally, the upper motor neuron controlled the lower motor neuron, causing unequal groups of muscles to be balanced. In spastic palsies, owing to the upper motor neuron lesion, there was an unbalanced action of muscles, and talipes equinus resulted. The orthopædic surgeon reasoned that if he could balance these muscles he could do the child good, therefore he knocked out part of the nerve supply which supplied the strong group, and if there was anatomical shortening of the strong group he lengthened the tendons of those muscles. If the balance of the muscles was restored by surgery, the result was often very good.

## Section for the Study of Disease in Children.<sup>1</sup>

President—Dr. ERIC PRITCHARD.

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### Case of Consanguinity.

By E. C. WILLIAMS, M.B.

THE parents of the patient are brother and sister. The child has a very poor circulation, large malformed ears, and is said to have attacks of violent temper at times, and to be very fond of gaudy clothes. The mentality also is much delayed.

### Case of Pulmonary Fibrosis following a Tuberculous Infection.

By E. C. WILLIAMS, M.B.

PATIENT, a girl, aged 15 years, is stunted in appearance, and her fingers and toes are extensively clubbed. The heart is drawn over to the right side, the apex beat being in the fifth right interspace just within the nipple line. There are well-marked physical signs of bronchiectasis, and a history of expectoration of large quantities of clear, foul-smelling liquid. She has been under treatment for four years at the Children's Hospital, keeping fairly well on creasote inhalations with internal administration of tincture of garlic and occasional courses of cod-liver oil and malt extract combined with phosphate of iron.

### Case exhibiting the Tooth-Marie-Charcot Type of Muscular Atrophy.

By E. C. WILLIAMS, M.B.

PATIENT, a boy, aged 12 years, was knocked down by a tramcar when 4 years old and this accident was followed by wasting of the muscles below the knees. This wasting is now well shown; there is also some talipes equinus and the small muscles of the thenar eminences of both hands are beginning to waste. With the exception of the loss of tendo-Achillis jerk on both sides the reflexes are normal, no sensory changes are present, and there is no sphincteric involvement. The boy keeps fairly well, and, with the aid of surgical iron supports for the legs and daily massage, can move about satisfactorily.

<sup>1</sup> Provincial Meeting of the Section held at Bristol, June 22 and 23, 1923.

### Three Cases of Congenital Syphilis.

By R. H. NORGATE, L.R.C.P.Lond., M.R.C.S.Eng.

*Case I.*—Patient, a girl, aged 5 years, who came under treatment for arthritis, the knee-joints being greatly enlarged and the legs rigidly flexed on the thighs. At the commencement of treatment there was an open ulcer on the dorsum of the left foot, and a presystolic murmur was audible at the cardiac apex. The patient had been confined to bed for many weeks before I saw her. A Wassermann test gave a positive result, and galyl injections resulted in a rapid improvement in all the conditions; but to complete the treatment it was necessary to apply extension methods to the legs. The patient can now walk about in quite a satisfactory way. The presystolic murmur is still audible.

*Case II.*—Sister of the first patient, showing skin lesions on face typical of congenital syphilis.

*Case III.*—A boy, aged 15 years, with sallow complexion and senile expression. Liver and spleen both enlarged. Heart: Apex beat in fifth interspace,  $\frac{1}{2}$  in. outside nipple line; a systolic murmur is heard over the whole of the precordium, and a well marked thrill present with maximum intensity over an oval space including the aortic and pulmonary valve areas. Wassermann reaction positive. Patient has had an intermittent temperature for several weeks. The case appears to be one with a specific congenital cardiac lesion and a superimposed endocarditis.

### Case of Ichthyosis and a Congenital Heart Lesion associated with a Small Pulse.

By O. C. M. DAVIS, M.D.

PATIENT, a boy, aged  $9\frac{1}{2}$  years. There is no obvious cardiac enlargement, but a systolic murmur over all the valve areas; also a thrill, both thrill and murmur having a maximum intensity over the aortic area.

### Three Cases exhibiting Mongolism.

By O. C. M. DAVIS M.D.

ONE of these patients, a boy, aged 2 years, also has a congenital heart lesion, there being a loud systolic murmur audible over the whole of the precordium. The other two patients, girls, aged 5 years and 10 years respectively, are affected with a somewhat uncommon congenital malformation of both ears; in both cases the concha auriculæ is completely divided into two cavities by the union of the crus helicis with the anti-helix.

**Case of Encephalitis Lethargica followed by Symptoms of Paralysis Agitans.**

By O. C. M. DAVIS, M.D.

PATIENT, a boy, who had an attack of encephalitis lethargica three years ago now showing after-effects of paralysis agitans type. He had a typical Parkinsonian expression, with considerable rigidity of both upper and lower extremities, exaggerated knee-jerks, and tremor of both hands. There appears to be some functional element present as the boy gave a vigorous demonstration with a skipping-rope, and immediately afterwards relapsed into his state of apathy.

**Case of Bronchial Asthma.**

By O. C. M. DAVIS, M.D.

PATIENT is a girl liable to severe attacks of bronchial asthma following indiscretion in matters of diet. She is much relieved by the administration of benzyl-benzoate.

**Case of Clubbing of Fingers and Toes.**

By O. C. M. DAVIS, M.D.

PATIENT, a girl, aged 17 years, has well-marked clubbing of fingers and toes, and is liable to severe attacks of dyspnoea. There is intense cyanosis, but no cardiac enlargement nor abnormal auscultatory phenomena. Erythrocytes, 9,500,000 per cubic millimetre. An electro-cardiogram, kindly taken by Dr. Carey Coombs, showed no particular preponderances. Probable diagnosis: Widely patent foramen ovale.

Dr. O. C. M. DAVIS also showed a girl, aged 3½ years, with Congenital Absence of the Iris in both Eyes.

**Acute Nasal Sinus Disease in Young Children.**

By E. WATSON-WILLIAMS, M.C., Ch.M., F.R.C.S.Ed.

IN approaching the subject of acute sinus disease in quite young children, say 3 years old and under, it is desirable to discuss only the conditions analogous to those seen in adults. In the new-born acute osteomyelitis of the maxilla occurs; the maxillary antrum at this age is a shallow groove, in a cancellous bone mainly occupied by the dental germs; its infection in all the twelve cases I have been able to collect appears an inconsiderable incident in a much more serious condition. This is mentioned, as these cases are often described as acute sinusitis especially on the Continent [1].



## ANATOMY OF THE MAXILLARY SINUS IN CHILDREN.

In very young children my own observations of sinus disease have been confined to the maxillary antrum, the only sinus that approaches the adult development. At birth only a shallow horizontal groove in the outer wall of the middle meatus, this sinus enlarges with the eruption of the milk teeth. Until, however, the second dentition begins it remains a small cavity, with a relatively wide mouth, and a floor above the level of the floor of the nose. To these conditions we may attribute the comparative infrequency at this age of sinus disease as a clinical entity. Doubtless acute infection occurs with every acute catarrh, but the factor of *retention* which plays an essential part in the development of sinus disease is usually lacking.

## FREQUENCY OF SINUS DISEASE IN CHILDREN.

But when the anatomical disposition favours retention, even partial, the child is no more immune to sinus disease than is the adult. Even in the adult few patients present themselves in the acute stage [2]; probably in the young child most cases fail to attract attention, though a purulent nasal discharge, especially unilateral, perhaps with headache, following immediately on a coryza, should make us suspicious. The majority of the cases will pass into the stage of chronic sinus disease. This condition in young children has only recently begun to receive attention. The earliest case I have found is one in a child aged 5 years, confirmed by exploratory puncture in 1915 [3]. Since 1919 the papers of Dean, Cleminson, P. Watson-Williams, and Mollison [4] (who all used, substantially, the technique introduced by Dr. P. Watson-Williams [6]), and a discussion at the Laryngological Section of the Royal Society of Medicine, have shown that chronic sinus disease in children—and so, as we must suppose, acute sinus disease—is by no means rare. The papers alone cover over 300 cases, in many of which attention was directed to the sinuses on account of polyarthritis or other severe complications: Of children requiring “tonsil and adenoid” operation, sinus infection was present in 15 per cent. (Dean) and 22 per cent. (Mollison).

## SEVERE ACUTE SINUS DISEASE.

Although we can, as a rule, only presume a past acute stage from discovering the chronic disease, occasionally, again as in the adult [5], the infection is so virulent as to produce swelling, redness, and tenderness of the cheek, with fever. The two cases shown to-day sufficiently illustrate the condition and course.

*Case I.*—R. C., male, aged 3 years. March 6, 1923: Brought up with swelling of right cheek of twelve hours' duration.

History: Child had a cold a week ago. Tonsil and adenoid operation four months ago, normal course.

Examination: Right cheek red and swollen, swelling extends from lower eyelid to edge of mandible, from ala of nose nearly to ear; central part of swelling tender, boggy, like an abscess about to point. Right nostril discharges thin, white, *offensive* pus; child good colour, slightly fractious. Temperature 99°6 F. Teeth appear normal.

Operation: Ether; right antral exploration with suction syringe through middle meatus; muco-pus was withdrawn, which, later, on culture, yielded streptococci. Left antral exploration; macroscopically clear return. The right antrum was opened by

means of rasps in the inferior meatus. The mucosa was not disturbed, except on the cut wall. The antral floor was slightly higher than the nasal floor. The extent of the cavity appeared to be rather over 1 cm. from front to back, rather under in the other diameters.

After-treatment: Daily lavage of antrum.

Course: Temperature came down the night following operation and remained down. Swelling had subsided by the fourth day. The discharge ceased during second week, and child left hospital on the twelfth day.

Questionnaire, after one month: Answer: "Perfectly well." Six weeks after leaving hospital right nasal discharge recurred; daily lavage of the antrum was again successful in clearing it up.

Present condition (three and a half months later): Quite normal.

*Case II.*—V. P., male, aged 8 years. April 4, 1923: Brought to hospital with swelling of the right cheek of two days' duration.

History: Child had a very bad cold last week; seemed very ill the night before admission; was better on admission. Nasal discharge reported.

Examination: Right cheek swollen, red and tender, the skin showing slight vesication, suggestive almost of erysipelas. Swelling extends from lower eyelid to  $\frac{1}{2}$  in. above mandible, from ala to nose to within 1 in. of ear; on palpation, the margin of swelling is found to be fairly definite. No nasal discharge seen. Temperature 99° F. Teeth normal. Tonsils normal, very small pad of adenoids.

Operation: Antral exploration under ether, with suction syringe through middle meatus. Right antral return fluid contained thick pus, which later, on culture, grew streptococci. Left antral return fluid macroscopically negative, but culture showed a streptococcal infection. The right antrum was opened in the inferior meatus, with rasps; mucosa not curetted. Cavity appeared rather less than 2 cm. in extent from front to back, and only a few millimetres—say 7 or 8—from side to side.

Treatment: Daily lavage of right antrum.

Course: Temperature normal the night following operation. Next day the skin had cleared up, and the swelling had disappeared by the third day. Discharged from hospital on April 13, 1923 (tenth day).

Questionnaire, after three weeks: Answer: "Perfectly well."

About June 14 (i.e., two months after leaving hospital), a small swelling of the right cheek occurred, and right nasal discharge recommenced. Further treatment will be necessary—probably sinus lavage. It is interesting to observe in this case the typical "adenoid" facies and to notice the fact that there is now bilateral discharge, worse on the left side.

#### DIAGNOSIS.

The diagnosis of acute sinus disease in children of this age will present some difficulty. The history of a recent cold, and the presence of a nasal discharge, especially unilateral discharge, call for investigation. Sneezing has been noted as a common symptom. Even in chronic disease there is very frequently no visible discharge. Signs of an abscess in the cheek (unless of dental origin) are pathognomonic. All observers agree that the results obtained by transillumination and skiagraphy are so uncertain as to render these methods valueless.

#### TREATMENT.

Confirmation of the diagnosis is obtained by exploratory puncture of the antrum. A general anæsthetic is necessary, and the exploration should be made through the middle meatus [6]. This obviates all risk of missing the antrum or of puncturing the orbit [4]. In an ordinary case lavage through the cannula will probably suffice. In the presence of manifest external signs of suppuration, I have not ventured to rest here. The antro-nasal wall is removed below the inferior turbinate by means of rasps. When this is done,

the small size and high floor of the cavity must be borne in mind, and care taken not to damage the outer wall, or to cut too far forward, for fear of damaging the teeth or dental germs. The mucosa is not curetted. The whole procedure takes about a minute. The aperture thus made will close in the course of a few weeks in most cases. It allows of daily lavage of the antrum for the short time this is necessary. The administration of cocaine is inadvisable at this age, but with gentleness no suffering need be caused.

The prognosis appears to be good so far as my observations have gone.

My thanks are due to Mr. J. P. I. Harty, F.R.C.S., for permission to use the notes of these cases.

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Dr. J. H. NIXON read a paper on "The Schick Reaction and Diphtheria Anaphylaxis."

Dr. O. C. M. DAVIS read a paper on "The Clinical Significance of certain Urinary Conditions in Childhood."

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SESSION 1922-23

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CLINICAL SECTION



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## **Clinical Section.**

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### **Traction Lesion of the Right Brachial Plexus, involving the Fifth and Sixth Groups.**

By A. E. MORTIMER WOOLF, F.R.C.S.

**HISTORY:** Patient, a male, aged 42, in April, 1922, was lifting a piano, with both hands extended, i.e., lifting from the floor. He felt "a lot of cords" pulling at the root of his neck, and a certain amount of pain down the arm as far as the elbow. One week later he noticed that the muscles of his arm were wasting, and this has become progressively worse.

**Present condition:** Marked wasting of deltoid, supraspinatus, infraspinatus. Supinator longus apparently not affected. Brachialis anticus probably involved. This muscle reacts to faradism, but none of the other involved muscles do so. They react to galvanism, but it takes a larger number of milliamperes than normally to cause contraction, especially so in the deltoid and supraspinatus. There is a slight loss of sensation of the skin over the deltoid muscle over a small area. There is no other loss of sensation of any kind. X-rays do not show any cervical rib.

### **Case of Bilateral Hammer Great Toes.**

By A. E. MORTIMER WOOLF, F.R.C.S.

**PATIENT,** a female. Double hammer toes involving both big toes. This has been noticed for many years, and is now causing her pain. X-rays do not show any bony deformities of the phalanges. No history of poliomyelitis, or of heredity.

### **Case of (?) Epithelioma of the Penis.**

By A. E. MORTIMER WOOLF, F.R.C.S.

**PATIENT,** a male, aged 52. First seen on account of a urethral fistula. His history was that four months previously a small abscess had developed on the dorsum of the penis, and had ruptured, and from that time urine had passed through it.

On examination a large indurated ulcer, about 1 in. in length, was seen projecting under the skin,  $\frac{3}{4}$  in. broad. The margins were extensively indurated, the whole of the corpora cavernosa apparently being involved, and urine was passing over the ulcer. An F.6 catheter was passed with considerable difficulty, but the obstruction seemed due to general thickening of the corpora cavernosa and not to any definite stricture.

There is a previous history of gonorrhœa but not of syphilis. In the three weeks since patient was first seen, the ulcer has extended on to the ventral surface of the penis, and the urine is now leaking out below as well as above.

The suggestion is that there may have been a gumma of the penis which has become malignant.

*Postscript.*—Since he was shown he has been admitted to hospital. Wassermann reaction negative. Three pieces were excised from the margin of the ulcer. Upon microscopical examination the sections showed a chronic inflammatory process, with no suggestion of malignant disease.

The skin has been slit up, and improvement is rapid. It is hoped to show the patient again at a subsequent meeting.

### Case of Multiple Exostosis and Hip Disease.

By C. E. SHATTOCK, M.S.Lond., F.R.C.S.Eng.

PATIENT, a boy, aged 4. Multiple exostoses. Has never been able to walk properly. Right hip movements very limited. X-ray shows small exostosis on neck of femur. Paternal grandfather and paternal uncle and father all have multiple exostoses.

*Subsequent Note.*—Hip was explored, and two small exostoses found on neck of femur. Synovial membrane pulpy. Microscopical examination of synovial membrane showed the lesion to be tuberculous.

### Case of Partial Gastrectomy for Cancer of the Stomach.

By R. P. ROWLANDS, M.S., F.R.C.S.

PATIENT, a male, aged 50, had attacks of gastritis eight months ago and since then has been ailing, with indigestion and vomiting at times. He has lost  $1\frac{1}{2}$  st. in weight. Lately he has vomited altered blood. X-ray examination showed deformity of the pyloric part of the stomach, with obstruction. Marked subacidity present. A movable tumour could be felt at the pylorus but no secondary growths anywhere.

Operation (July 24, 1922): A large growth was found in the pyloric segment with several malignant glands above and below the pylorus. The carcinoma was adherent to the neck of the pancreas and transverse mesocolon so that portions of these had to be removed. Otherwise the removal was fairly easy, about half the stomach being taken away with 1 in. of duodenum, and an anastomosis made in front of the colon after the Polya-Mayo method. The jejunal opening was begun only 4 in. below the duodenum. The loop did not appear to press upon the colon.

Patient has gained 2 st. in weight.

## Case of Myeloma of the Outer Condyle of the Femur, showing the Result of Bone-grafting.

By ALAN H. TODD, M.S.

PATIENT, a male, aged 24, was carrying a basketful of loaves down a flight of steps, on June 17, 1921, when he slipped and fell; he states definitely that he twisted his knee in falling. His knee filled with fluid, which persisted for several weeks. In the course of the next six months he had several "attacks," as he calls them, in which his knee gave way suddenly, and caused him to fall, or nearly to fall; after each attack a certain amount of synovitis occurred, and some pain and tenderness were present. The tenderness was localized to the region of the external semilunar cartilage. When first seen, in December, 1921, there was no grating or clicking in the joint, and fluid was absent. The joint could be flexed to  $80^{\circ}$  and extended to  $170^{\circ}$ . The right thigh was 1 in. smaller than the left.

The history and general symptomatology strongly suggested an injury of the external semilunar fibro-cartilage, but a routine radiogram revealed the presence of a large myeloma in the outer condyle and part of the shaft of the femur.

Operation (January 5, 1922): The limb was exsanguinated with a Martin's bandage, and an Esmarch's tourniquet was applied. The soft tumour-mass was curetted and wiped out of the cavity, the outer wall of the femur being found to be almost completely destroyed. The articular cartilage of the femur was intact, though the bone inside it was all but gone, so that only a few glistening scales of bone remained adherent to its upper surface, and the cartilage was transparent. Every particle of the tumour was carefully removed, and the cavity was then swabbed with pure phenol, followed by alcohol and ether. An incision was now made along the iliac crest, from the anterior superior spine backwards, and the muscles on either side were peeled back, subperiosteally. Two saw-cuts, 3 in. long and 3 in. apart, were then made into the ilium, and the base of the square thus marked out was fractured, and the piece of bone was removed. It was split, so as to expose the cancellous bone within, and then cut into pieces of various sizes, which were packed as tightly as possible into the myeloma-cavity. The pieces were then impacted one upon another, and driven into firm contact with the walls of the cavity, by hammering, so that the fragments of bone might, if possible, obtain nutrition at an early date, and also move about as little as possible. The limb was put up for the first fortnight in a Thomas's bed-calliper, and then in a plaster, which the patient wore for three months. The wound healed by primary intention, and there was no serous or other discharge.

Microscopical examination of the tumour by Dr. G. W. Nicholson revealed a typical myeloma.

At the end of three months, a weight-relieving calliper was applied, and the patient began to walk. Care was taken that the splint was slightly longer than the limb, so that the heel of his foot was never touching the heel of his boot; thus the weight was transmitted through the steels and not through the lower limb.

Subsequent radiograms show the gradual consolidation of all the grafts. It is interesting to note, not only the gradual consolidation of the transplanted bone, but also the gradual remodelling of the outline of the outer edge of the femur, along the line of weight-transmission.

#### 4 Todd: *Myeloma*; Cassidy: *Patent Interventricular Septum*

The man is able to extend his knee fully, and to flex it to well under a right angle. (The photographs show a range of movement of  $120^{\circ}$ .) No grating is present, and he has no pain. He can stand and walk a little without the calliper, but has not yet been encouraged to do so. There is no knock-knee, and the right thigh is now only  $\frac{1}{2}$  in. smaller than the left. It is proposed that the splint should be worn for a year from the time of operation.

The large gap made in the ilium has caused no discomfort or disability, and is gradually filling up with a partly-ossified mass of fibrous tissue. The use of bone from this source commended itself to me on the following grounds: (1) A large quantity was needed, and could be readily obtained; (2) it is better, on general principles, to use cancellous bone to replace cancellous bone; (3) the bone could be conveniently obtained, without moving the patient, and no special tools were necessary for the purpose; (4) far greater shock would have been caused by the prolonged hammering and chiselling that would have been necessary in order to obtain so large an amount of compact bone from the shaft of the tibia, whilst if rib had been used, a large amount of time would have been spent, and the material obtained would have been less suitable.

It is generally agreed that amputation is not necessary in cases of myeloma, and that efficient curettage will ensure the saving of life, but in cases of myeloma of the femur amputation has often been performed in the past, in the idea that gross deformity might otherwise occur, or else that conservative measures might involve an undue expenditure of time and prolongation of crippledom. This case shows that this is not a fact. Amputation for myeloma of a condyle must now be regarded as totally impermissible, and obsolete.

One little point deserves notice. The calliper used has been a weight-relieving one; that is to say, it has always been longer than the man's limb, and his heel has never touched the heel of his boot, so that no weight can have gone through his limb. Nevertheless, the latest radiogram shows the slightest possible compression of the articular surface of the outer condyle. This can only be due to the unopposed pull of the thigh-muscles, which have dragged the tibia tightly up against the condyles. If I ever meet a case of this kind again, I shall use the old-fashioned Thomas's knee splint, which is made with a screw-traction attached to the heel of the boot, so as to maintain active extension on the leg and stretching of the thigh; there will then be no compression of the condyle at all.

It was fortunate that the articular cartilage had escaped perforation and that the knee-joint was intact. Had the interior of the joint been involved, I should still have avoided amputation, by excising the knee and filling the interior of the myeloma cavity with bone-grafts. The patella, denuded of its outer sheath of compact bone, makes a very useful graft with which to fill a large part of the cavity in these instances.

#### Case of Patent Interventricular Septum.

By M. A. CASSIDY, M.D.

PATIENT, an army pensioner, aged 47, was blown up and gassed in May, 1915. He states that he was unconscious for six days, and on recovery suffered from facial paralysis and loss of memory. A cardiac murmur was discovered for the first time when he was in hospital in 1915. He now complains of faintness, vertigo, and insomnia.

He says that he played half-back for Dulwich College, and has been through nine campaigns—as a result of which he wears seven medals with eighteen bars. Before 1915, he had undergone repeated medical examinations, and has always been passed as fit for general service. He had a slight attack of rheumatic fever in 1895, but apart from this, has always been perfectly healthy.

On examination, a loud, harsh, systolic murmur is heard, associated with a thrill, both of maximal intensity, in the fourth space 1 in. outside the left sternal edge. The apex beat is in the sixth space in the left nipple line. X-ray examination reveals an enlargement of the heart, chiefly on the left side. The Wassermann reaction is negative. There is a trace of cyanosis but no clubbing. Blood pressure: 170 systolic, 100 diastolic.

*Remarks.*—The physical signs seem to point conclusively towards a patent inter-ventricular septum, but it is difficult to believe that this condition can have been congenital, since it is almost incredible that a murmur of this intensity should not have been discovered before 1915. It seems likely that the congenital defect may have been a weak area with, possibly, an aneurysmal bulging of the septum, which was ruptured as a result of the explosion in 1915. An instance of traumatic rupture of the ventricular septum has been recorded by McOscar and Voelcker.<sup>1</sup>

### Cases of Duodenal Ulcer to illustrate Certain Points in Diagnosis.

By H. S. SOUTTAR, C.B.E., F.R.C.S.

#### (I) HIGH ACIDITY AND DUODENAL ULCER ASSOCIATED WITH PTOSIS. RELIEF WITH GASTRO-ENTEROSTOMY.

PATIENT, a male, a tailor, aged 43. For eighteen years has had attacks of pain after food, with intervals of freedom. Duration of attacks and of intervals very irregular. An attack began early in July and lasted until admission to hospital in August. During the attack the pain was in the left epigastrium, piercing in character, coming on two hours after meals, and awakening him at 2 a.m. Occasionally he has vomited with relief.

Test meal showed HCl 0.22 per cent.; total acidity, 69. Bismuth meal showed ptosis and some distension of the duodenum, suggesting an ulcer.

On August 22 operated upon; an indurated ulcer was found on the anterior surface of the first part of the duodenum. It was invaginated, and a posterior gastro-enterostomy was performed.

Patient made an uninterrupted recovery.

#### (II) VISCEROPTOSIS WITH LOW ACIDITY AND DUODENAL ULCER.

PATIENT, a male, packer, aged 49. For three years has had attacks of pain after food, with intervals of freedom. The attacks last about two weeks, the intervals have varied from two to twelve months. His last attack began a month before his admission to hospital.

He has pain in the epigastrium about one and a half hours after food. It is not relieved by food, and does not awaken him at night. Occasionally he has vomited with relief.

<sup>1</sup> *Trans. Path. Soc. Lond.*, 1897, xlviii, p. 47.



The test meal showed HCl 0.06 per cent. ; total acidity, 35. Bismuth meal showed marked ptosis of the stomach with poor tone.

Operation, September 6, marked visceroptosis found. In the first part of the duodenum ulcers were found on the anterior and posterior walls, facing one another. The anterior wall was invaginated, and anterior gastro-enterostomy was performed.

Patient made an uninterrupted recovery.

(III) DUODENAL ULCER INVOLVING PYLORUS; CHANGE IN EPOCH OF PAIN;  
RELIEF BY GASTRO-ENTEROSTOMY.

PATIENT, a labourer, aged 48, for six years had attacks of pain in the right epigastrium, with intervals of partial relief. The pain came on two hours after taking food, and sometimes kept him awake at night; frequently vomited, with relief resulting. Recently the pain has become more severe, and for about six weeks has come on much earlier, even as soon as half an hour after eating.

Test meal showed HCl 0.12; total acidity, 50. Bismuth meal showed distension of duodenum, rapid emptying of stomach, and irregularity of pylorus.

Operation on September 6: a chronic ulcer of the duodenum was found. An old scar extended for 1 in. from the pylorus, in the margin of which a small crater could be clearly felt, the peritoneal surface showing signs of more recent inflammations. The old scar was invaginated, and a posterior gastro-enterostomy performed.

## Clinical Section.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

*Meeting at London Hospital, Friday, November 10, 1922.*

Held on the invitation of the Academic Units.

THE following cases were shown:—

- (1) *By the Medical Unit.*—Pneumo-hæmothorax, Pulmonary Osteo-arthritis, Carcinoma of Stomach, Infective Endocarditis, Diplegia with Bilateral Choreo-athetotic Movements in a Boy aged 13, Addison's Disease, Sarcoma of Lung, Pernicious Anæmia.
- (2) *By the Surgical Unit.*—Plastic Operations on Face (Three Cases), Carcinoma of Femur, Multiple Pneumococcal Arthritis, Operations for Undescended Testicle, Acute Pancreatitis, Perforation of Meckel's Diverticulum, Duodenal Ulcer in a Girl, aged 21.

### Sciatica in a Woman suffering from Lead Poisoning.

By BERNARD MYERS, C.M.G., M.D.

MRS. J. R., aged 39, was admitted to the Royal Waterloo Hospital during November, 1921, complaining of a swelling over the sacrum and right loin, great pain in her right leg, and severe headache.

Tender area found in rectum, apparently connected with the external swelling, the pain of which was worse in the morning but much less noticeable towards the afternoon. Patient looked distinctly ill and anxious.

The pain in the right leg was due to a severe attack of sciatica. The right thigh was  $\frac{1}{2}$  in. smaller than the left. The knee-jerks were increased; there was some ankle-clonus. The big toe response was doubtful extension. There was no wasting of the calf muscles. The left leg showed nothing abnormal, but the knee-jerks were a little increased; the cranial nerves were not affected. Dr. Bickerton examined the eyes, and stated that there was nothing abnormal in the discs, &c., and no ocular paralysis. The liver was a little enlarged; the spleen could not be felt. The colon was loaded.

(Examination of the blood by Dr. Leatham, on December 21, showed the hæmoglobin to be 100 per cent., the red blood cells 5,840,000, leucocytes 12,400, differential count normal. No abnormal changes in cells noted.)

History: Patient had been doing war service as a painter for two and a half years previously. Her health, previously good, had become greatly affected, and she suffered from lead colic, bluish-black line on her gums, albuminuria, and a double wrist- and foot-drop. These conditions were no

## 8 Myers: *Sciatica in a Woman suffering from Lead Poisoning*

longer present, but she complained of severe headache. Urine now normal. Tendency to constipation. Periods were regular. Had apparently ceased work when taken ill and had received some treatment.

The swelling over the sacrum and loin was regarded as probably due to angio-neurotic oedema. Lead poisoning considered the most probable cause of the symptoms.

Treatment: Rest in bed; 5 gr. doses of iodide of potassium, thrice daily, and a drachm of sulphate of magnesium every morning. A lessening of the symptoms was soon noted.

Dr. H. G. Archer detected traces of lead in her urine and blood after exhibition of the iodide.

Her bed being wanted, patient was treated as an out-patient, but improvement was only partial, and she was readmitted to hospital last March. Although the sciatica had nearly disappeared and she was able to walk very slowly with a distinct limp, there was marked pain around the right hip. She had not suffered any further from swelling over the sacrum, or pain in the bowel. The headache had not quite gone, but was much less than formerly.

It was decided to inject turpentine subcutaneously at the outer side of the right thigh. This was followed by a severe local inflammation with rise of temperature to 101° F. Slight superficial incisions were made into the inflamed area on the third day after the injection, and a fair amount of serum exuded, but no pus was detected. After being ten days in bed she was allowed up, and was now able to walk without pain, the first time for many months; a week later she walked without any difficulty whatever.

The patient now enjoys excellent health and has put on weight, making up for the weight previously lost before treatment. The headaches have disappeared: there is still a slight liability to constipation. She has been quite free from pain in the leg and hip for over six months, and she can walk normally.

The iodide mixture was continued until April of this year. This doubtless was of distinct use, but the turpentine injection seemed to be the determining factor in finally eradicating the pain.

## Clinical Section.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### Case of Aneurysm of the Arch of the Aorta and Innominate Artery in a Woman.

By BERNARD MYERS, C.M.G., M.D.

MRS. B., then aged 54, was admitted to the Royal Waterloo Hospital in May, 1918, for abdominal pain and constipation. A cystic ovarian tumour was found and extirpated during the following month. She returned to hospital in September, 1920, complaining of pain between the shoulders and also in the right shoulder. She also felt giddy. A double aortic murmur was discovered. There was some throbbing in the vessels of the neck and she was sent into the ward for observation in January, 1921. There seemed to be a little dilatation of the transverse arch of the aorta, although the X-ray report stated that the aorta was of normal size. The Wassermann reaction was positive and she was given a course of glucose-galyl and mercury and potassium iodide. The dilatation of the arch of the aorta became more pronounced in the course of a few weeks and a diagnosis of aneurysm was made. The aneurysm has continued to increase in size until at the present time it extends to about  $\frac{3}{4}$  in. above the manubrium sterni. The innominate artery seems also to have become aneurysmal and this condition apparently affects to some extent the common carotid and subclavian; there is evidently now some pressure exerted upon the cords of the right brachial plexus. The apex beat is in the sixth space just outside the left nipple line. The right pulse seems sometimes to be a little smaller than the left.

In April, 1921, the right pupil was noted to be smaller than the left, and there was also a sweating of the right side of the face especially upon pressing the tissues on the right side of the neck. Slight tracheal tugging is palpable. Salivation had become a great nuisance to her but this was controlled by the exhibition of tincture of belladonna. Pain in the right shoulder and front of chest was now greatly disturbing her. The muscles of the right shoulder and arm had become a little atrophied and also a little weak. Pain is felt on the outer side of the right shoulder and to the outer side of the upper forearm, and also in the fourth and fifth fingers. Over most of the inner side of the right arm there is a numb feeling. Lately she has complained much of a boring pain  $1\frac{1}{2}$  in. to the right of the sternum over the second rib, where a pulsation is felt. A similar pain is felt 1 in. to the right of the spinous process of the fifth dorsal vertebra. She complains at present of a feeling inside her chest as if something were going to burst. Râles are generally present at the bases of both lungs, but, so far, the air entry into each lung appears to be quite free. There have been no pressure symptoms on important veins or the vagus nerve so far, but swallowing has occasionally been difficult.

[December 8, 1922.]

**Treatment:** In addition to the anti-syphilitic course, the main indication has been to deal with the pain and sleeplessness caused through it, and also to treat her constipation. To relieve the latter she has had *mist. alba* and *cascara*. Potassium iodide has been continued, salicylates have been tried for the pain, and tinct. of hyoscyamus and opium; also belladonna plaster externally, but all to no effect. Aspirin, paraldehyde and trional have proved equally inefficacious in the removal of the sleeplessness or pain. Presumably the question of operation cannot be considered.

It has been interesting to observe the gradual transition in growth of the aneurysm from a slight dilatation of the aorta to its present large size and of the change from the not particularly marked symptoms to the constant pain and distress now evident. I am indebted to Dr. John Parkinson for having kindly had an X-ray made of the chest, which shows the condition.

The opinion of Members of the Section would be greatly appreciated with regard to any treatment likely to alleviate the pain.

In the discussion on the case the use of morphia for the relief of the pain was recommended.

### Case of Purpura Hæmorrhagica.

By BERNARD MYERS, C.M.G., M.D.

MRS. D., aged 25, admitted an in-patient to the Royal Waterloo Hospital on September 30, 1921, suffering from purpura hæmorrhagica, had been a patient in this hospital twelve years previously on account of bleeding from the nose and mouth, with blood spots and bruises, evidently purpura hæmorrhagica. Upon the present occasion she was found to be bleeding from the upper and lower gums and vagina, and to have many hæmorrhagic spots on the face, the right eye, limbs, and body generally. There were two ecchymoses. She was feeling distinctly weak. So far she did not look very anæmic. Fourteen days previously the patient had been delivered of her first baby, which was breast fed.

On October 4, a catheter specimen of urine was found to contain a fair quantity of blood. The next day bleeding was seen to be taking place from the uterus also. The hæmorrhages from the mucous membranes and skin already mentioned were increasing. There was a slight improvement until October 17, when a large clot the size of a large hen's egg was passed from the vagina. She looked paler and felt very weak. The following day the vagina had to be packed by Dr. Barris, who had previously removed an offensive smelling blood-clot containing some membrane. Another offensive smelling blood-clot weighing 2 oz. was also removed from the uterus; 1 c.c. of pituitrin was then administered and sensitized streptococcus vaccine given in a 50 million dose, repeated in twenty-four hours by 100 million, and forty-eight hours by 200 million.

The cultures from the catheter specimen of urine showed saprophytic bacilli with a few colonies of *Staphylococcus albus*, both of which Dr. Leatham thought were accidental. There was no growth of streptococci or of *Bacillus coli*. On October 21, the swab from the uterine cavity contained streptococcus, but it was entirely overgrown by *Bacillus coli*.

The temperature was mostly normal or slightly above normal in the morning and was raised from half to three degrees in the evening; but at the time of the large hæmorrhage towards the middle of October the temperature

went up to 104° F. and remained higher than usual for five days. After the administration of human blood the temperature was generally less elevated, and about a week later came down to normal level. The respirations never exceeded 25 per minute, but the pulse became rapid whenever the hæmorrhage from the mucous membranes was in excess.

Treatment: Absolute rest in bed was enjoined, milk diet given and a mixture of calcium lactate 10 gr. administered daily. This, however, had no appreciable effect upon the hæmorrhage. Adrenalin solution 1 in 1,000 was applied to the bleeding surfaces of the gums with some effect; 10 c.c. horse serum were given on several days, but did not control the hæmorrhage. Among other things tried were injections of pituitrin (1 c.c.) as previously stated, anti-streptococcic serum (10 c.c.), hæmostatic serum (2 c.c.), but none of them checked the bleeding. Ultimately transfusion of human blood was carried out on October 21, by Dr. T. Joeke from a No. 4 donor; 120 c.c. of blood were mixed with sodium citrate solution so as to make 0.25 per cent. of the citrate, and injected into the median basilic vein. The result was most satisfactory, and within twenty-four hours there was appreciable diminution of hæmorrhage from the mucous membranes and no fresh purpuric spots developed on the skin. Recovery after this was gradual and uninterrupted. No fresh hæmorrhage was observed on the skin a week later and all bleeding from the mucous membranes had ceased. The patient's colour improved and she felt a little stronger. She was then given collosol iron 10 minims in  $\frac{1}{2}$  oz. of water thrice daily after food.

The patient has remained exceedingly well since discharge from hospital with the exception of a slight attack of purpura during July this year. She came to hospital immediately it was noted, and the administration of calcium lactate 10 gr. thrice daily seemed to be all that was necessary to prevent further occurrence of hæmorrhages on the skin or mucous membrane.

Although the small amount of membrane which was found in the vaginal blood-clot might possibly have had something to do with the onset of the condition, it would appear that this was a primary case. Vaccine and sera were comparatively useless in arresting the hæmorrhage, but the success of transfusion of human blood was marked and of the first importance.

#### BLOOD COUNTS.

November 7: Red cells, 3,330,000; hæmoglobin, 60 per cent.; leucocytes, 8,000.

November 21: Red cells, 4,200,000; hæmoglobin, 80 per cent.

November 25: Red cells, 4,100,000; hæmoglobin, 80 per cent.; leucocytes, 10,000, of which the polymorphonuclears were 72.6 per cent.; lymphocytes, 21 per cent.; large mononuclears, 2.8 per cent.; transitional, 2 per cent.; eosinophils, 1.6 per cent.

No blood platelets were seen.

### Case of Adiposis Dolorosa.

By BERNARD MYERS, C.M.G.. M.D.

MRS. H., aged 45, was shown at this Section last year as a case of adiposis dolorosa.<sup>1</sup> Since then she has continued to improve greatly in health so long

<sup>1</sup> *Proceedings*, 1921-22, xv (Clin. Sect.), p. 6.

as she was taking pituitary gland. Whenever this was discontinued for a short period she invariably experienced general weakness within a few days, and this weakness lasted until pituitary gland was again administered. The dose given has been 1 gr. of the whole gland twice daily after food. For a time thyroid gland 1 gr., once daily, was also administered, and this apparently helped to reduce her weight from 18 to 15 st., but the drug responsible for her general well-being, increased strength and energy is the pituitary (whole gland). The menses, which were scanty and irregular before treatment, became regular and practically normal after the exhibition of pituitary.

Dr. Herbert Williamson, Senior Gynæcologist, St. Bartholomew's Hospital, became interested in this patient and she was placed under his charge, at St. Bartholomew's, for several weeks. During her stay there the sugar tolerance test was repeated and found to be practically identical with the test previously taken, the report of which is summarized as follows: Blood-sugar before taking glucose amounted to 0.142 per cent.; one hour after 50 gr. of glucose had been taken it had increased to 0.314 per cent.; and at the end of two hours it was 0.183 per cent. Glucose was absent from the urine both before and after the administration.

Dr. R. G. Canti, Pathological Department, St. Bartholomew's Hospital, kindly examined the section which was taken from the largest painful nodule on the left leg. His report states: "The section shows skin and subcutaneous tissue. Beneath the latter is a considerable thickness of fat. The lobules of fat are separated by dense strands of fibrous tissue. The fibrous tissue appears to be actively proliferating, large numbers of fibroblasts being present and large numbers of small blood-vessels. Patches of small round-celled infiltration are to be seen here and there, especially in the neighbourhood of the vessels. Endarteritis is present, the larger vessels having thickened walls due to proliferation of the intima, and some of the smaller ones are obliterated. No nerve tissue is to be seen."

There has been a tendency for the nodules to diminish or disappear from the areas where previously found on the arms and for new ones to appear in the legs. Where these nodules have disappeared, pigmented areas not infrequently remain on their sites.

In February of this year (1922), the following blood-count was made: Hæmoglobin, 100 per cent.; red cells, 5,420,000; leucocytes, 7,800; polymorphonuclears, 59 per cent.; lymphocytes, 32 per cent.; large mononuclears, 3 per cent.; transitional, 4 per cent.; eosinophils, 2 per cent.

### Case of Thrombo-angeitis Obliterans.

By GEOFFREY EVANS, M.D.

PATIENT, a male, aged 42, tinsmith by occupation. His mother was an Englishwoman, his father, a Jew, and so far as he knows his family has been in England for a hundred years. Has been under my observation since February of this year (1922). He was first admitted to St. Bartholomew's Hospital on account of pain in the left big toe and of waves of unpleasant sensation passing upwards from the toe to the head. He also complained of headaches and of irregular action of the heart.

History: His complaint—recurrent attacks of gangrene of the toe—dates back to an influenza illness twenty-two years ago. The first attack occurred



in March, and succeeding attacks, recurring every two to three years, have generally begun in spring and usually last two to six months. The duration of the present attack, however, has been three years.

An "attack" consists in the appearance of a bleb or blister, often provoked by "slight accidental injury," on one of the toe-joints or terminal phalanx of the great toe or about the nail of one of the other toes. This bleb, sometimes described by the patient as a corn, sometimes appearing as a small ischæmic area, breaks down, suppurates, is fomented and finally heals. In succeeding attacks the necrotic process has extended more deeply and formed a perforating ulcer and necrosis of bone, as a result of which one or more phalanges have had to be amputated at different times from three toes in either foot.

No general symptoms accompanied these attacks until after operative treatment in 1921 (September) involving scraping of the terminal phalanx of the left big toe. General symptoms of blood poisoning set in, with fever lasting fourteen days. After recovery there was a feeling of numbness in both lower limbs brought on by walking. At other times the patient complained of a "bursting feeling" in the left big toe when the leg was left in a dependent posture, the pain being relieved by raising the foot. This sensation lasted for a few seconds, and was followed by a feeling of coldness in the foot. The foot would then feel normal again, and in a few seconds the patient would have a sensation of warmth and swelling in the knee. The same sensation would travel to the head, and this was followed by a hot burning feeling, spreading inwards and upwards from the trunk to the back of the head and neck and passing down the arms. As this sensation travelled through the chest he complained of a sensation of pins and needles in the heart, and the heart's action seemed to the patient to become irregular. At the same time he would feel as if his head were going to burst. Such attacks are followed by a feeling of complete exhaustion, and they would end by the patient falling into a light sleep, from which he awakened cold and sweating.

Habits: The patient smokes fifteen cigarettes daily, and drinks an occasional glass of beer. Although his work involves soldering, there have been no symptoms suggesting lead poisoning. In 1916 he was passed C3 for the Army, but did not serve owing to his being in a scheduled occupation.

Past History: Shingles at the age of 12. No other illnesses. There is no history of sore throats or scarlet fever, and no history of venereal disease. He is said to have coughed up some bright blood in 1920: there is no other suggestion of lung trouble.

Family History: His father is alive and well at the age of 72. His mother died, aged 55, from pneumonia. He has one sister, aged 40, who has lung trouble, and who has a son, who has been treated for pulmonary tuberculosis.

Present condition (February, 1922): Fairly healthy, of slight build, complexion rather pale and sallow. Blood-count shows no anæmia nor variation from the normal. Widespread gingivitis and pyorrhœa alveolaris. Dental films show considerable absorption of the inter-dental alveolus of all the teeth, and periodontitis of the right upper canine; no apical rarefaction (Dr. Finzi). Tonsils slightly swollen, not definitely infected, and no affection of the tonsillar lymphatic glands. Thorax, poor expansion. Heart and lungs, normal. Abdomen normal. No signs of disease of the central or peripheral nervous system. Urine normal. No indican. Retinæ normal. Blood: Wassermann reaction negative; after provocative novarsenobillon also negative. Streptococcal complement-fixation test (Dr. Kenneth Stone, June 21, 1922): serum gave a weak positive reaction with a compound *Streptococcus salivarius* antigen;

reaction negative with *Streptococcus pyogenes* and *fæcalis*. Electro-cardiogram normal. Skin histamine reaction less than normal (Dr. Mackenzie Wallis).

Upper extremities: Neither right nor left radial pulses palpable. Radial arteries just palpable; do not feel thickened. Ulnar pulses palpable, vessels not thickened. Blood-pressure: 140/76 mm. Hg. The skin of the hands and fingers seemed atrophic, surface somewhat glazed, colour pink, warm to touch.

Lower extremities: Dorsalis pedis pulses not palpable. Arteries not palpable: weak pulsation in the posterior tibial arteries. Radiograms of the limb arteries show no calcification. The skin of the toes and feet is somewhat shiny and flushed, with a slight degree of cyanosis. On the inner and under aspect of the left big toe there is a small depressed scar. The last two phalanges of the second and third left toes have been amputated. There are five small scars over the second and third metatarsals. The terminal phalanx of the fifth toe and two of the distal phalanges of the fourth toe have been amputated. The toe-nail only of the third is missing. The feet are warm.

Progress of the Case: The patient was knocked off smoking and became an abstainer. He was given thyroid extract, long periods of rest in bed, massage, and exercises for the lower limbs. No improvement occurred, and after six months' observation, on account of the persistence of the attacks of very unpleasant sensation (already described) originating in the terminal phalanx of the left great toe, it was finally decided to amputate this toe at the patient's urgent request. This operation was done by Mr. Roberts.

On the opinion that the condition from which the patient was suffering was a true endarteritis due to the action of a toxin absorbed from some focus in his body, and the only focus of infection discovered being his teeth, it was ultimately decided that all of them should be extracted. This was done in October. The patient's condition does not appear to have changed since he first came under observation in February.

*Microscopical Examination of the Amputated Toe.*—One digital artery was normal, the other showed complete obliteration. There was no increase in the adventitia nor was there perivascular infiltration. The media appeared somewhat atrophied, the nuclei staining less well, and the muscle cells were apparently fewer in number than in the normal artery. The intima was replaced by a hyaline mass completely blocking the lumen. This mass was permeated by strands of fibrous tissue, and was practically acellular.

Clinically, the case appeared to be one of thrombo-angeitis obliterans. The histological observations, on the other hand, do not support this diagnosis, although they do not definitely negative it.

### Case of Thrombo-angeitis Obliterans.

By ST. J. D. BUXTON, F.R.C.S.

E., MALE, aged 40, an engineer, attended out-patient department at King's College Hospital in January, 1922, complaining of cramp pains in both legs when walking. He was treated as a case of intermittent claudication for six months with little improvement.

Past history: Scarlet fever at 12 years. Passed into the Army A1. Married ten years. One healthy child. Not a Jew. Not a heavy cigarette smoker.

In summer, 1922, pain in the sole of the right foot was severe. The foot was then reddish-blue, slightly swollen, but no ulceration was present. Arterial pulsation could not be felt around the right ankle, but the left anterior tibial artery was pulsating. His general condition was good, although he was thin. Sir Charlton Briscoe, under whose care the patient had been, said that there was no sign of organic disease of the central nervous system. Urine normal, and blood Wassermann reaction negative.

In August he developed a whitlow round the right little toe nail, and a perforating ulcer under the middle of the transverse arch. These showed no reaction to any treatment.

In October he was put to bed. Three teeth were extracted for superficial caries, and others scaled. Urine normal, and Wassermann reaction negative to blood and cerebro-spinal fluid. Blood-pressure 120 mm. Hg in right arm. It could not be taken in the leg, as no pulsation could now be felt at either ankle, nor heard with a stethoscope. Gangrene was commencing round the ulcer on the sole.

He was given pot. and sod. iodide and liq. hydr. perchlor. for some weeks, with no sign of improvement.

In November the foot became more swollen, red and shiny; there was gangrene the size of a half-crown on the sole of the foot, and the flexor tendon was exposed. The third toe was involved.

November 15: Loss of part of the limb was inevitable, and it was determined to try the effect of sympathectomy, Sampson Handley's modification being the method employed. The femoral artery was exposed in Hunter's canal. It was small and very feebly pulsating. Five minims of 80 per cent. absolute alcohol were injected into the adventitia. Skin temperatures were then taken of the feet, and although the temperature of each foot was the same at the end of the operation, this temperature did not remain constant, the right foot becoming colder again, as before the operation.

November 18: The right foot was less swollen, but the gangrene had spread over half an inch more tissue.

November 22: No further spread, and the gangrene is drier and the foot not swollen.

November 29: No further spread. Amputation was performed through the middle of the leg.

December 10: The wound healed satisfactorily.

#### MICROSCOPICAL REPORT.

Sections of arteries, nerves and muscles were cut from the limb. Just above the ankle the antero-tibial artery (*see* photomicrograph, p. 16) shows a very small lumen, which is lined with endothelium. Between this and the elastic layer there is a great proliferation of the intima, but the elastic layer is intact. New blood-vessels are present among the proliferating cells; these have thick walls and small lumina. There is a cellular infiltration in and around the adventitia. These changes are less marked in the upper part of the artery, and are present to a less degree in the plantar and post-tibial arteries. The nerves are normal, and their arteries appear normal. The muscles show no abnormal changes, but the vessels in the foot show proliferation of the intima.

Dr. F. PARKES WEBER said that in a Hebrew man with typical *angeitis obliterans* of both lower extremities he had been able to observe the onset of *angeitis obliterans* of one radial artery at the wrist. It commenced as a nodular inflammatory swelling,

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and gradually the swelling subsided, leaving the radial artery at the wrist permanently pulseless. This was, in fact, an attack of "nodular" thrombo-arteritis, similar to the attacks of superficial nodular thrombo-phlebitis in the leg, from which the patient had likewise occasionally suffered. The family history in that case was very remarkable, and the patient's granddaughter, aged 12 months, had recently been in hospital under Dr. Weber's care, suffering from recurrent attacks of typical Raynaud's syndrome, from which she first commenced to suffer at the age of 8 months. Dr. Weber believed that the affected arteries in cases of thrombo-angeitis obliterans were sometimes originally hypoplastic (too small in proportion to the size of the limb).



Anterior tibial artery, showing thrombo-angeitis obliterans. Intimal proliferation is very evident, with vascularization of this tissue. ( $\times 47$ .)

### Case of Scorbutic Infantilism.

By MAURICE CASSIDY, M.D.

PATIENT, a male, T. W., aged 18.

Family history: The patient's elder brother is aged 19 and his voice has not yet broken: his younger brother died when aged 18 months and is described as being "very backward." He has three sisters, all normally developed as also are his parents and all his other relatives. His mother is now in hospital with cancer of the breast.

Past history: Full-time child, born naturally. Teeth erupted at the normal time. Bottle-fed, as were all his brothers and sisters. Has lived all his life in Yeovil, Somerset. Has never eaten either fruit or vegetables, as he says they made him vomit. Has subsisted largely on bread and butter and cakes, but often has meat, eggs, bacon, porridge, and fish, but takes no vegetables except potatoes. Drinks milk only with tea or cocoa.

He has always been undersized, but was in other respects considered a healthy and active child till the age of 13, when it was noticed that his gums were swollen and that pus was discharged from them. When he was 15 years

old his mother noticed that he began to "walk on his toes," and subsequently he gradually lost the power of walking, and became bed-ridden.

History of present condition: In March, 1921, when aged 16, he came under the care of Dr. H. R. Unwin, of Yeovil, to whom I am indebted for the foregoing history. He had severe pyorrhœa, and there were hæmorrhagic swellings in the muscles of the calf and in the hamstrings, first of the right, later of the left leg. The knee-joints were swollen and doughy. A diagnosis of scurvy was made. All his teeth were extracted and he was put on an anti-scorbutic diet and given intramuscular injections of sodium cacodylate. The hæmoglobin percentage was then 50, and there was a marked lymphocytosis. The legs became painful and, in spite of applications of splints and extensions, contractions of the legs appeared.

Since leaving the hospital at Yeovil a year ago, he has lived at home, receiving no treatment except occasional massage, and he has reverted to his favourite anti-vegetarian diet. His legs have become increasingly painful and tender, the slightest attempt to move or even touch him making him scream. He has also complained of "rheumatic pains" in the arms between the shoulder and elbow, and has been slightly tender there. About four months ago his legs and feet began to swell. He was admitted to St. Thomas's Hospital on November 3, 1922.

Present condition: His appearance is that of a boy about 12. The voice has not broken, the external genitalia are small but not rudimentary; there is no hair on the pubes, nor on the face. The head is small and round, and the scalp is covered with fine, silky, dark hair. The cheeks are chubby, and on admission were bright red, so also was the tip of the nose; the complexion now (December 8) is more waxy. There is a little brown pigmentation on the forehead, especially marked over a crescentic area above the right eyebrow.

Mentally, the patient is intelligent and alert, though somewhat childish. His weight is 3 st. 7 lb.

The upper limbs are remarkably thin, and the triceps muscles appear to be particularly atrophic. There is some limitation of abduction at both shoulder-joints, but apart from this there is full range of movement and the power is good for the size of the muscles. The heads of the radii are enlarged, but not tender, nor are the heads of the humeri tender, though there is a little diffuse tenderness to palpation all over the upper arm.

Lower limbs: Both knees are fixed in flexion, the right to  $45^{\circ}$ , the left to  $90^{\circ}$ . The right hip is everted  $90^{\circ}$ , flexed and abducted, and the left hip is internally rotated  $90^{\circ}$  and adducted  $25^{\circ}$ . The patient is just able to move his feet and toes, but otherwise the limbs are powerless. When the limbs were examined under anaesthesia by Mr. Rowley Bristow, only a few degrees of passive movement at the knees and hips were possible; the fixation of the joints appeared to be due to synovial and ligamentous thickening rather than to muscular spasm, though even under anaesthesia the muscles felt hard and brawny. At time of admission, the legs were very œdematous, especially below the knees, where the skin was extraordinarily tense and glossy. Under treatment this œdema has almost disappeared and the skin is now desquamating. There was marked swelling of the knee-joints, this being apparently due to bony and synovial thickening. The lower ends of the femora were exquisitely tender, the lightest touch or movement causing the patient to scream.

Reflexes: Knee-jerks just present. Triceps, supinator, and Achilles jerks and plantar reflexes all normal.

Sensation: Everywhere normal.



FIG. 1.—Opacities in lower end of radii and in epiphysis of terminal phalanx of right little finger.

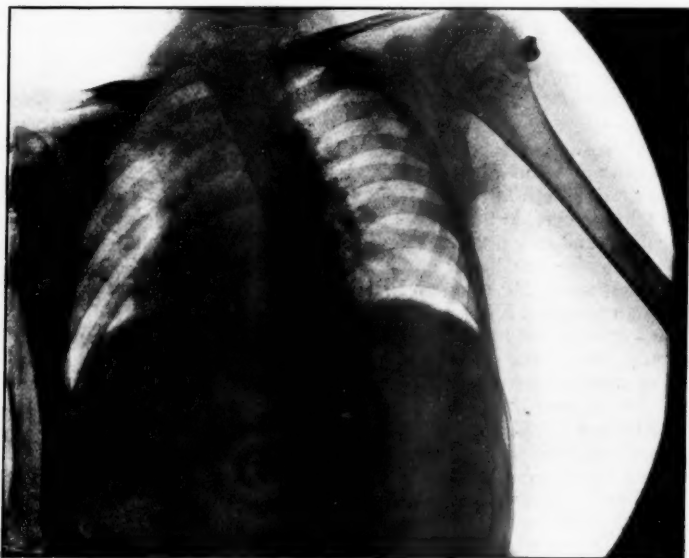


FIG. 2.—Extreme rarefaction of shaft of humerus and opacity in its head.

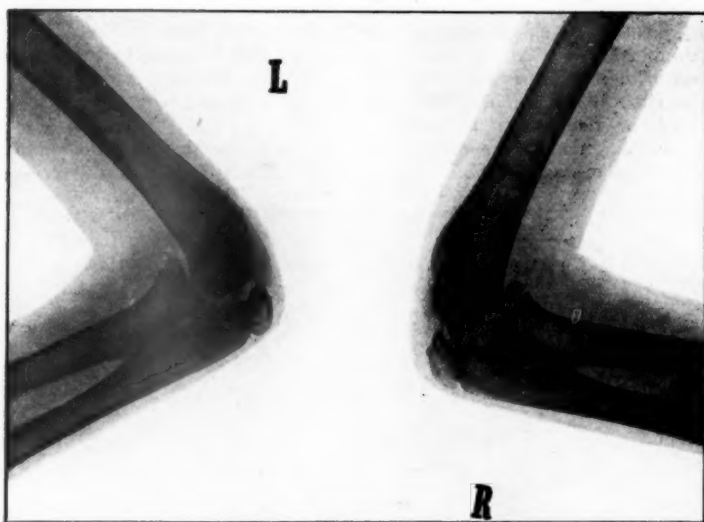


FIG. 3.—The opacities seen near the shoulder and wrist-joints are not present near the elbow-joints.

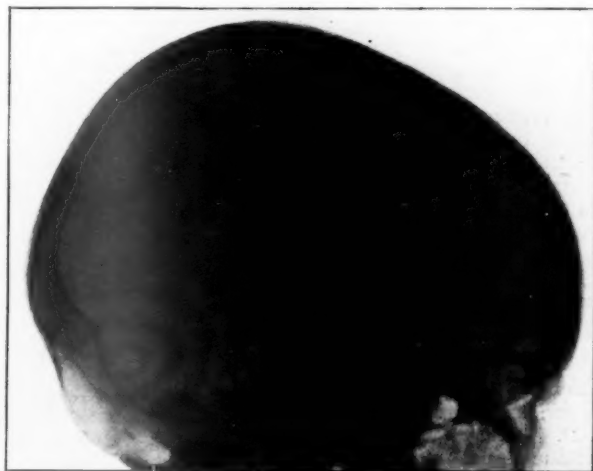


FIG. 4.—No gross abnormality detected; the sella turcica is normal.



Bladder and rectum : There has been occasional incontinence of urine and faeces, but this seems to have been accidental.

Trunk : Marked dorsal scoliosis, concave to left. Dilated cutaneous veins present over the upper part of the chest in front. Slight, but definite, beading of the ribs. Abdomen not distended; its wall is resistant to palpation and none of the viscera can be felt.

X-ray examination revealed extraordinary rarefaction of all the bones of the skeleton except the skull (figs. 2, 5, 6). There are remarkable conical,

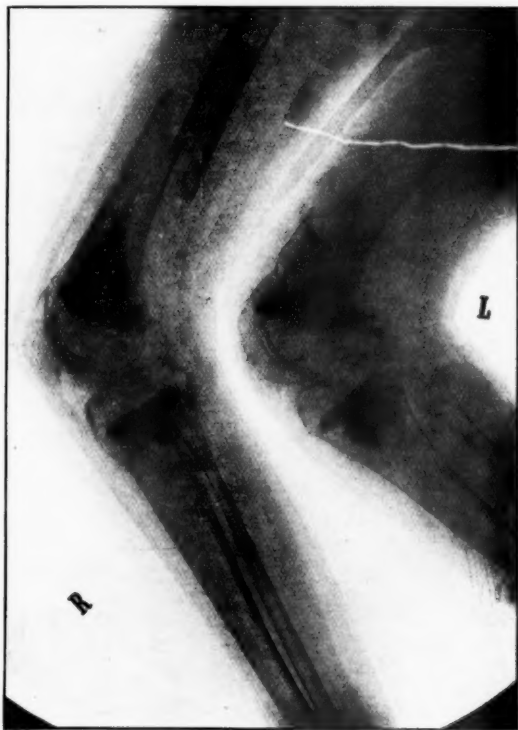


FIG. 5.—Recent spontaneous fracture of right femur. Less recent fracture of left femur. Opacities in lower ends of diaphyses of femora, and in upper ends of diaphyses of tibiae. Extraordinary rarefaction of shafts of all bones.

structureless opacities in the ends of the diaphyses of the long bones (figs. 1, 5, 6), most evident on each side of the knee-joints; not present at the elbow-joint (fig. 3). Recent fracture present at junction of lower and middle thirds of right femur and an older fracture at lower end of diaphysis of the left femur (fig. 5). No history of anything to account for these fractures. Epiphyses have not united. Terminal epiphysis of right little finger opaque and structureless. Sir Archibald Reid reports that the radiological findings in this case are, in his experience, unique.

Basal metabolic rate + 0.9 per cent., i.e., normal (Dr. Gardiner-Hill).  
Wassermann and Sachs-Gorgi reactions negative.

## BLOOD COUNT.

Erythrocytes	...	...	...	4,038,000 per cubic millimetre
Hæmoglobin	...	...	...	56 per cent.
Colour index	...	...	...	0.7 per cent.
Leucocytes	...	...	...	No obvious leucocytosis or leucopenia
Poikilocytosis	...	...	...	+ + +

## Stained Blood.

Polynuclear neutrophils	...	...	...	...	52.5 per cent.
Small lymphocytes	...	...	...	...	25 "
Large "	...	...	...	...	14 "
Large hyaline cells	...	...	...	...	5.5 "
Coarsely granular basophilic cells	...	...	...	...	3 "
Coagulation time	...	...	...	...	1.46 "
(Normal control	...	...	...	...	2.14)

November 21, 1922.

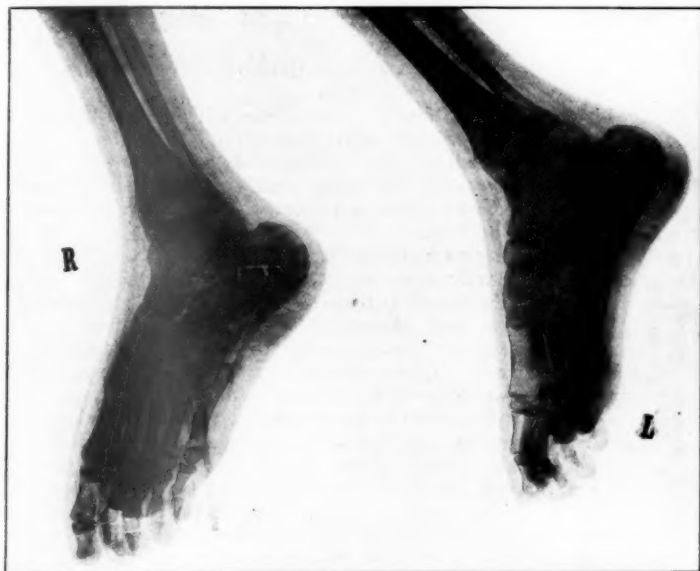


FIG. 6.—Opacities in lower ends of diaphyses of tibiae. Great rarefaction elsewhere.

Renal efficiency: Dr. de Wesselow reports as follows: Blood urea, 19 mg. per 100 c.c.; plasma inorganic phosphorus, 2.97 mg. per 100 c.c. Urea concentration test: first hour, 2.2 per cent., 186 c.c.; second hour, 3.12 per cent., 80 c.c.; third hour, 3.07 per cent., 96 c.c. Urine: Protein *nil*, sugar *nil*. The renal efficiency from the point of view of nitrogenous excretion is excellent. The plasma inorganic phosphorus (phosphates as P) is at an average normal figure. As far as this goes it is against any rickety element in the disease.

November 22, 1922: Calcium of serum, 9.5 mg. per 100 c.c. (normal 9.6 to 10.6—no appreciable reduction).

Blood-pressure: 120, systolic; 80, diastolic. There was evening fever of between 100° and 101° F. for ten days after admission, but this has now subsided.

Treatment and progress: Since he still refuses fruit and vegetables he has been given lemon, orange, and cabbage juice flavoured with peppermint, as a medicine. He also takes cod-liver oil and marmite. He has had very gentle massage of the legs. Under these measures his pain and tenderness rapidly disappeared, and the œdema has almost gone. Mr. Bristow proposes to correct the deformity of his hip-joints under anaesthesia and to put him up in a plaster bed, and subsequently to straighten the knees. I am also considering giving light treatment to the legs. The recent fracture of the right femur seems now to be firmly united, clinically.

### Case of Acromegaly in a Girl aged 16, with Congenital Heart Disease (Aortic Stenosis).

By E. STOLKIND, M.D.

FAMILY history: The patient's parents are of average height. The mother suffers from neurasthenia and headaches. (A doctor advised her twelve years ago to have all her teeth extracted as a cure for the headaches; and this advice was followed.) The father suffers from headaches and also from eye trouble. The patient is the youngest of their three children. The eldest sister, aged 19, is healthy.

The patient at birth was a big baby, weighing about 11 lb. She was bottle-fed, and began to walk when about 2 years. The doctors (specialist) had already diagnosed "congenital heart disease." When she was 6 years old, she had scarlet fever, and when aged about 7 she had measles. From 5 to 13 years of age she suffered from nocturnal enuresis, but after that less frequently. Her menses began a year ago at the age of 15; at first every two months, and later every month for two days and in slight quantity.

There are all the symptoms of aortic stenosis: her pulse is about 70 and regular. Blood-pressure, 140 to 90 (R. R.). The apex beat and dullness of the heart are about half an inch to the left of the nipple line. There is a systolic thrill, and a rough, loud systolic murmur in the aorta (less audible at the apex), conducted to the carotid arteries. The second sound of the aorta is slightly accentuated. X-ray examination shows that the heart is slightly enlarged downwards and to the left, with a rounded apex. It seems that there is a slight increase in width of the aortic shadow (for a girl of her age).

The patient left school at the age of 14, as a "backward girl." She began to grow fast during the last seven years; her growth has been very rapid throughout the last two years. She is now 5 ft. 5.9 in. high and weighs 9 st. 7½ lb. The length of her feet is 10¾ in., and of the middle fingers of her hands 4¾ in. She is taller than her parents and her elder sister.

There is some polyphagia, but no polydipsia or polyuria. No sugar or albumin in urine.

The patient is very weak, and soon becomes tired after exertion. She often drops things which she holds in her hands. She is very shaky and trembles, and has been stooping for some months past. She is restless in bed at night.

During the day she cannot sit quietly, and keeps moving her legs, arms, &c. She has no memory; she is mentally slow, and takes no interest in anything.

The superciliary areas are abnormally developed. The lower jaw is visibly prognathic.

Dr. Findlay's X-ray examination shows that the posterior clinoid processes of the sella turcica, which are rather large for a girl of her age, appear to end in a fragment of bone hanging backwards. According to Dr. J. H. D. Webster the patient shows, on X-ray examination, a sella turcica, large, but within normal limits of size; the posterior clinoid processes exhibit backward extending processes, which constitute a normal variation at the tentorial attachments. The nasal sinuses are large, and there are slight signs of acromegalic changes in the supra-orbital ridge and mandible. The X-ray signs are those of acromegalic gigantism, which has not yet reached the stage of sellar malformation.

The eyes were tested by Mr. Lindsay Rea and were found normal.

Treatment: X-ray applications.

*Remarks.*—This is a very rare case: a combination of acromegaly in a girl aged 16—juvenile acromegaly—with congenital aortic stenosis. Congenital aortic stenosis is also very rare.<sup>1</sup>

### Case of Calcinosis.

By Professor F. LANGMEAD, M.D.

(ABSTRACT.)<sup>2</sup>

PATIENT, a female, L. D., aged 35. Scattered through the subcutaneous tissue, especially of the extremities, are small, hard, shotty nodules. For the most part they are freely movable, and are tender when touched or pressed upon by clothes. They are aggregated most thickly on the posterior surfaces of the forearms and on the shins. These were first noticed by the patient, accidentally, in 1916, and, according to her story, fresh nodules have continued to appear ever since; several of them being present both in the arms and the legs within six months of the onset.

On the fronts of the knees and on the elbows, the skin hangs in folds, and in these situations and also on the knuckles hæmorrhages frequently occur, as the result of pressure or a slight injury. The skin generally is thin and ill-nourished, perhaps no more so than in correspondence with the general state of nutrition of the patient. There is no scleroderma.

The hæmorrhages first appeared in 1919. She suffers greatly from cold hands and feet and chilblains in cold weather, and these parts are always cold and blue. Attacks of more definite cyanosis and cramp-like pains occur from time to time. Blood-pressure: Systolic 120, diastolic 70. During the time she has been under observation, since 1919, there have been occasional attacks of acute arthritis in the knees. Chemical examination of the nodules shows that they consist of organic material, with a small amount of calcium and magnesium phosphates and a trace of iron. Radiograms give clear pictures of the situations and sizes of the nodules, and show the presence of a group of calcareous glands in the mediastinum.

<sup>1</sup> E. Stolkind, "Hereditary Syphilitic Aortitis," *Brit. Journ. Child. Dis.*, 1920, xvii, p. 126.

<sup>2</sup> This case will be published in full elsewhere.

*Remarks.*—The case is a definite example of the disorder which has been called calcinosis and also petrification—names which have been given to the formation of subcutaneous calcified nodules. In many of such cases, scleroderma and sclerodactylia have been present, but these are absent in this case. In a few cases, fibrosis of muscle and arthritis have been recorded. The hæmorrhages in the case shown to-day are of considerable interest, and suggest that small blood-vessels are affected in the same way as the subcutaneous tissue. From microscopical preparations which have been made in two cases, in the first by Thibierge and Weissenbach, and in the second by F. Parkes Weber, it appears that the calcification takes place in pre-formed fibrous tissue which has undergone degenerative change. The occurrence of symptoms like those of Raynaud's disease has been described in cases previously reported, and may be explained either by compression of the peripheral vessels by the fibrous tissue or by the existence of changes in the vessel walls, similar to those found in the subcutaneous areolar tissue.

### Hydronephrosis of a Single Kidney; Spontaneous Rupture into the Peritoneal Cavity.

By PHILIP TURNER, M.S.

PATIENT, a male, H. L. C., aged 39, was admitted into Guy's Hospital on January 6, 1915, for inability to pass urine and for acute abdominal pain. There was no history of any preceding urinary or abdominal trouble. The onset of the pain was sudden. He was taken ill during the night before admission. A little blood-stained urine was passed when the pain started but none had been passed since. The pain commenced on the right side but soon became general: it had been severe and throbbing in character. Patient stated that he was able to feel two lumps on the right side, where the pain was first noticed.

On admission: The patient was in a condition of collapse. The abdomen was slightly distended: there was little respiratory movement and it was rigid and tender, especially on the right side. There were signs of fluid in the peritoneal cavity. A catheter was passed and 4 oz. of blood-stained urine were drawn off.

Operation: Immediate operation was decided upon. The abdomen was opened by an incision in the right semilunar line as, when the patient was anaesthetized, a distinct bulging could be seen, and a flaccid tumour could be felt, filling the right loin. On opening the peritoneum a great quantity of blood-stained fluid escaped. A dishful of this was saved for subsequent examination, which showed that it was urine mixed with blood. The tumour was found to be a ruptured hydronephrosis. A large tube was inserted into this and a second tube was employed to drain the peritoneal cavity.

Progress: The patient recovered well from the operation. After a few days suppuration occurred in the hydronephrosis and large quantities of urine and pus were discharged through the tube. On a few occasions a little urine was passed *per urethram*, but generally speaking the whole of the urine was passed through the tube. A week after the operation it was noted that about 8 oz. of urine and pus were passed by the urethra but the passage of urine by the normal channel then ceased. There was pyrexia of the septic type and the condition of the patient was not satisfactory.

An X-ray examination failed to reveal any calculus and gave no information as to the cause of the trouble.

Second operation: On January 29 it was decided to perform an exploratory operation with a view to ascertaining the exact condition and to relieve it if possible. The abdomen was opened in the mid-line and the urinary organs examined. No left kidney could be felt. Accordingly attention was directed to improving the drainage of the right kidney. No cause was found to explain the obstruction of the right ureter. After this operation there was violent vomiting and the wound gaped.

From this time the patient's condition slowly improved: the temperature gradually settled down and the pus in the urine diminished, and on his discharge, some five months later, the urine was free from both pus and albumin. From time to time a little urine escaped by the urethra but practically the whole amount was passed through the tube in the loin. Patient was discharged with the urine draining away into a rubber receptacle.

Patient was decidedly alcoholic and frankly attributes his trouble to drinking neat whisky. The evening before his illness he drank a whole bottle of "Red Seal," undiluted, and was naturally unable to give a clear account of what happened afterwards. No history of accident or injury could be obtained, but, of course, in his condition it might very well have happened. Some doubt must therefore remain as to whether the trouble was really spontaneous.

Present condition: All urine still escapes through the tube in the loin and is collected in a rubber urinal. There is a ventral hernia, doubtless the result of the opening of the wound during severe vomiting. The hernia is satisfactorily controlled by the belt which he always wears. His general health is quite good. The blood-urea content is about double the normal. He says that a little fluid escapes occasionally from the urethra but the nature of this is doubtful.

### **Case of Ulcerating Granuloma of the Pudenda in which Healing commenced immediately subsequent to the Administration of Antimony.**

By PHILIP MANSON-BAHR, D.S.O., M.D.

ULCERATING granuloma is a somewhat rare venereal disease occurring in certain tropical countries, notably in India, China, the West Coast of Africa, South America, and Northern Australia.

The present patient was originally infected in Hong-Kong in September, 1921: a small pimple appeared on the corona on its dorsal aspect five days after connexion. Gradually this ulcerated and spread backwards along the dorsum till it reached the root of the penis and the mons veneris, thence it has continued to spread, partly by direct extension, and partly by contact of apposing surfaces.

On the voyage home and subsequently he was treated as if the infection had been one of ordinary syphilis. From September, 1921, to October, 1922, he had been treated with six injections of neo-kharsivan, a similar number of mercury injections, potassium iodide, mercury inunctions, by scraping, by the application of pure carbolic and seven exposures to the X-rays, but without the least beneficial effect, for the ulceration continued to spread.

When first seen on October 21, 1922, there was deep ulceration of the skin of the root of the penis on the dorsum and on each lateral aspect; the ulceration itself was extremely deep over the symphysis pubis and there were

two isolated patches on the lower part of the abdomen. There was at the same time no cuticular covering to the dorsum of the penis, which was replaced by granulation tissue. The glans penis could not now be recognized except by the opening of the urethra at its extremity. The edges of the ulcers themselves were sharply-cut and undermined, and the bases were covered with particularly exuberant granulations encrusted with a yellowish plastic exudate. A noticeable feature was the absence of induration of the edge or base of the lesions. At the same time there was lymphatic œdema of the scrotum, but a total absence of involvement of the lymphatic glands. The Wassermann reaction taken on several occasions has been consistently negative.

Portions of the edge of the ulceration have been excised and sectioned for the presence of the peculiar parasitic bodies first described by Aragao and Vianna, but neither in them nor in smears have any such structures been seen. These organisms are now thought to be *Bacillus mucosus capsulatus*.

The interest of the case to us is not only the diagnosis, but the extraordinary specific response there has been to antimony treatment. The patient has now had 10½ gr. of antimony tartrate injected intravenously and seven intramuscular injections of oscol stibium with the result now seen; the hollows have become completely filled with healthy granulation tissue and the epithelium is spreading inwards with great rapidity, and soon the greater part of the damage will have been repaired. As far as the therapeutics are concerned, it is interesting to note that improvement seems to have been just as rapid after the intramuscular as after the intravenous injections of antimony.

The differential diagnosis of this condition is, of course, of great importance, and I would draw your attention to its resemblance to the soft venereal serpiginous ulceration often seen in this country (*ulcus molle serpiginosum*). It would be interesting to try the effect of antimony injections in this latter condition or indeed in other forms of chronic intractable ulceration.



## Clinical Section.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### Tumour removed from the Brain of a Child, aged 12.

By H. S. SOUTTAR, C.B.E., F.R.C.S.

THE tumour exhibited, which was recently removed, is an endothelioma which arose from the falx cerebri and was burrowing into the right frontal lobe.

The child (a patient of Dr. Cecil Wall) had a history of illness lasting fifteen months, during which the chief features were attacks of frontal headaches, vomiting, and a sensation of numbness in the left leg, followed by twitching, which spread up the leg, involving the left arm and face.

The tumour was removed across the substance of the right frontal lobe, through which an incision had to be made, the skull having been opened by means of an osteoplastic flap.

She made a perfect recovery, the temporary paralysis of the left arm and leg clearing up in ten days.

### Case of Chondroma of a Phalanx in the Hand.

By ST. J. D. BUXTON, F.R.C.S.

PATIENT, a male, aged 18, states that he "fractured" the first phalanx of the index finger of his right hand two and a half years ago, when he was boxing. In December, 1922, when boxing, he had another injury to the same finger, and came to hospital because he had a painful swelling on the finger.

Present condition: There is a swelling on the radial side of the base of the index finger of the right hand. The skin is stretched over it, and is not adherent. The swelling feels firm, attached to the bone, but not of bony hardness. It does not pulsate and egg-shell crackling has not been felt. The edges slope gradually to the bone, but not beyond either joint. There is slight limitation of flexion of the metacarpo-phalangeal joint. The swelling has remained the size of a hedge-sparrow's egg for the last month.

X-ray appearances: Absorption of base of the first phalanx on radial side, with slight expansion of the bone at the edge above and below the tumour. Some sclerosis along edge of absorbed area on one surface.

January 13, operation: Tumour enucleated from the bone without difficulty.

*Postscript.*—Microscopic examination shows the tumour to be a chondroma.

The points of interest in the case are the following: (1) The tumour was thought to be a myeloma, and was shown as such at the Section; (2) the tumour appears to have followed a definite injury.

### Case of *Cysticercus Cellulosæ*.

By VINCENT COATES, M.D.

H. G., MALE, aged 30, formerly schoolmaster. Onset of disease, May, 1917.

Past history: ? Slight heat stroke, malaria, dysentery, sand-fly fever, colic, and tonsillitis in India and Mesopotamia. A history of "fits," in which he loses consciousness for one to two hours. Does not bite tongue or lose control of sphincters.

History of present illness: Wounded 1917, and while convalescent noticed a lump in left shoulder in May, 1917. This was followed by a crop of lumps on his back, and later many other lumps appeared in various parts of his body. Many have disappeared, and he is sure that some have moved from their original sites. Gives a history of eating bad pork in India, but has never had a worm so far as he knows. His appetite used to be enormous, but he did not put on weight.

Examined first, June, 1921, when mobile tense swellings were felt in various parts of the body, some in the muscles and others less deep. Some were tender. Blood count showed 14 per cent. eosinophilia. No abnormality was found in the central nervous system. A lump was removed by Mr. Rendle Short, and proved to be a cyst. This was sent to Professor Walker Hall and to Professor Yorke, who diagnosed *cysticercus cellulosæ*. Many stools were examined before and after vermifuges, but no ova or segments were seen. No fits were noted in hospital, and after frequent repeated examination of central nervous system patient was discharged. The cerebro-spinal fluid was normal and there was no eosinophilia.

Patient was examined again in December, 1922. No organic disease was discovered, although he is stated to have had several fits lately. Some of the cysts have disappeared. There are no new cysts, and it would appear that several have definitely moved from their original position. The eosinophil count is 8 per cent.

The points of special interest consist in the mobility of the cysts and the fits of (?) functional origin. Does the absence of eosinophilia in the spinal fluid negative central nervous system involvement?

### Case of *Acholuric Jaundice*.

By VINCENT COATES, M.D.

F. J. S., MALE, aged 28, labourer by occupation. Duration of disease: (?) Since 1915.

History: Patient states that he has been jaundiced since he was in Gallipoli in 1915, when he had dysentery. He was sent to Egypt, and came home in 1916. Suffered from malaria in Palestine 1917. States that he suffers generally from weakness, and that he gets attacks of "wind," stomach-ache with pain between the shoulders, and shivering, accompanied by increase of jaundice and sometimes itching of skin. At these times his stools are light and his urine dark.

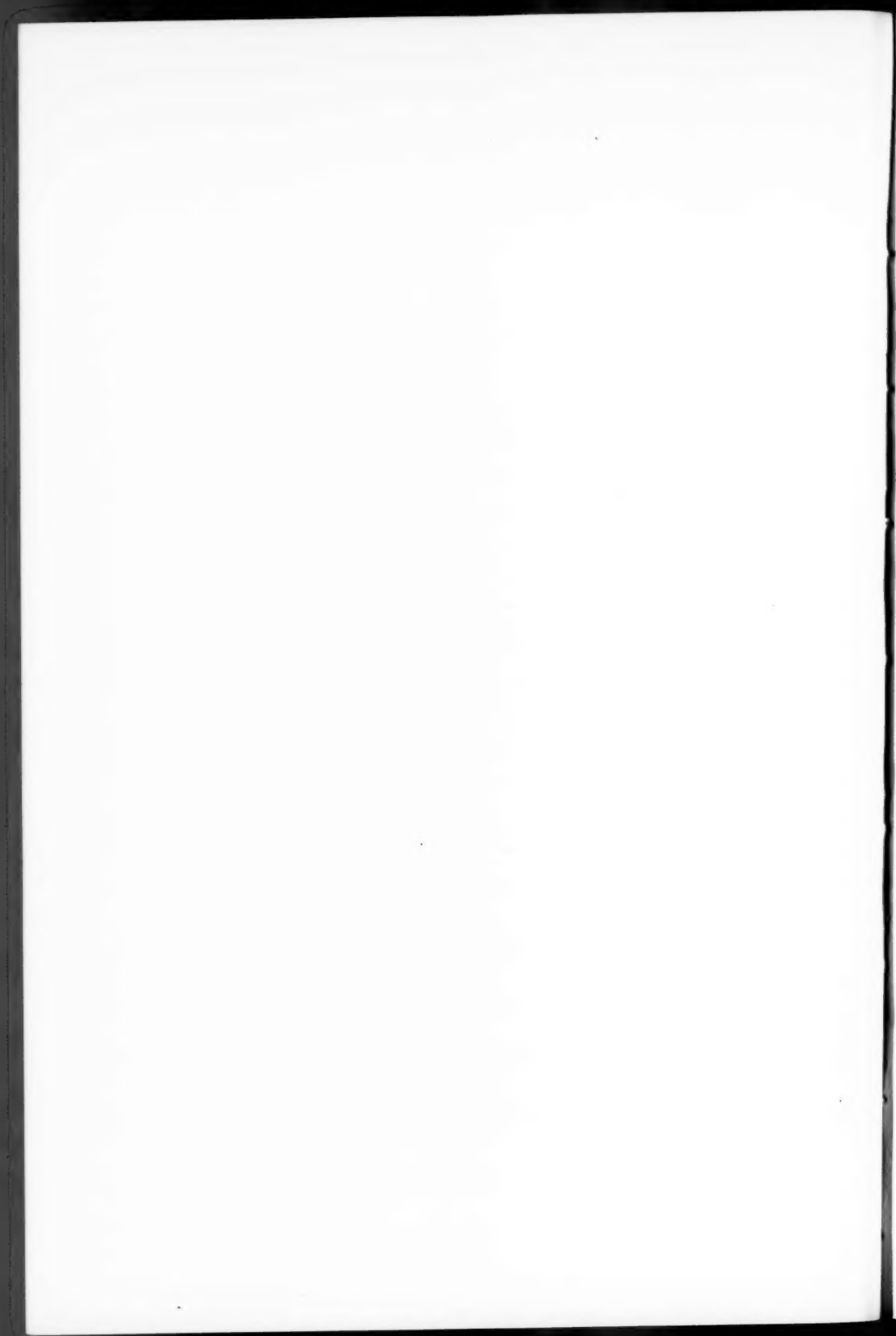
On examination in June, 1922, the following points were noted: Jaundice of skin and mucous membranes, no bile in urine. Slight enlargement of heart. Liver was palpable. Spleen palpable and mobile. Notch at umbilicus and lower pole three fingers from iliac crest. Stools repeatedly negative to bacterial and protozoal dysenteric organisms, also to enteric group. Wassermann reaction negative. Full blood counts made on half a dozen occasions, and the averages were then: Red cells, 4 to 5 million; white cells, 6,000 to 8,000; hæmoglobin, 80 per cent; colour index, once above 1 with microcytes, macrocytes and one normoblast.

For a few days preceding the occasion of the raising of the colour index, he had a slightly raised temperature; there was a trace of bile in his urine and his jaundice was deepened.

*Fragility of Red Cells.*

		0.7		0.6		0.5		0.4	
August—	Patient	...	—	...	—	...	+	...	+
	Control	...	—	...	—	...	—	...	±
September—	Patient	...	—	...	±	...	±	...	+
	Control	...	—	...	—	...	—	...	—

The points of interest in the case centre in the ætiology, and the question of operation as to whether removal of the spleen should be done or not.



## Clinical Section.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### Hirschsprung's Disease ; Partial Relief following Plication of the Sigmoid Flexure.

By W. G. SPENCER, M.S.

PATIENT, a boy, now aged  $7\frac{1}{2}$  years, the seventh child, born at full term, weighing 9 lb., was breast-fed for ten months, and progressed until a year old. Wasting ensued ; after measles at the age of 3 he developed blood-stained loose motions, rectal prolapse and undue distension of the abdomen with a poor appetite. He was first admitted to the Westminster Hospital for medical treatment at the age of  $4\frac{1}{2}$  years, with other characteristics of the disease ; his weight was 27 lb. and his girth at the umbilicus 22 in. By X-ray examination it was noted that delay in the movement of the intestinal contents commenced in the first part of the ascending colon. Under medical treatment the rectal prolapse ceased and his weight increased to  $31\frac{1}{2}$  lb. After discharge he relapsed and was re-admitted six weeks later. Following a second period of temporary relief under medical in-patient treatment there was a further relapse. He was re-admitted on the third occasion for surgical treatment owing to signs of subacute intestinal obstruction including vomiting. He weighed 29 lb. and his abdominal girth was 21 in. On May 13, 1921, an abdominal incision exposed somewhat distended but otherwise normal small intestines. These being collected and swung over to the right, there was exposed a greatly dilated and elongated sigmoid loop, twisted half a turn on itself and occupying the right epigastric region under the liver. The wall was much thickened, starting from the junction with the rectum ; the thickening of the wall and the dilatation of the lumen continued in a diminishing degree throughout the descending and transverse colon, but there was no marked elongation above the sigmoid flexure ; in addition to the great dilatation and thickening of its wall, the sigmoid loop appeared about three times its normal length. The application of sutures reduced its dilatation and elongation until the general appearance resembled that of a sausage. During the operation there was a small rapid pulse, shallow respiration and dilatation of the pupils, countered by keeping the head low and by saline infusions. Two months after the operation, under daily massage, he could take a full diet and his motions were regular. His weight had increased to 40 lb., the abdominal girth continuing at 21 in. He has now been under his mother's care at home for a year and a half. He continues stunted in growth, is pale, thin, has a poor appetite ; he has two or three loose motions in the morning, and one again at night ; the abdominal girth is about the same ; weight is 35 lb. The rectal prolapse and vomiting have not recurred. Improvement in weight and regularity of motions would doubtless follow readmission and treatment adapted to his individual requirements. This would be an essential preliminary if the only radical treatment, that of excision as performed by Sir William Arbuthnot Lane, were to be recommended.

Dr. F. PARKES WEBER said that in the case of a boy, aged 4, with Hirschsprung's disease, now in hospital under his care, a disagreeable complication was frequent involuntary passage of feces during sleep. (There was tendency to blocking of the rectum with hard fecal masses and there was obvious blunting of rectal irritability.) In a boy, aged 13, seen by Dr. Weber in 1904, acute ulcerative colitis supervened as a terminal fatal complication. Dr. Weber did not think that rectal prolapse (as in Mr. Spencer's case) was a recognized complication of Hirschsprung's disease.

*Postscript* (March 24, 1923).—After a further course in the hospital ward the boy's weight increased to 40 lb., and he had generally but one formed motion daily.

### Case of Myositis Ossificans exhibiting Acute Symptoms.

By C. MAX PAGE, D.S.O., F.R.C.S.

A. H. J., MALE, aged 28. This case appears to be one of myositis ossificans with typical X-ray appearances in the lower part of the right humerus. Its interest lies in the recurrent acute attacks of an inflammatory nature which have involved the whole of the extremity affected.

The man was wounded in the right hand on October 28, 1917; this wound was superficial and healed normally and gave rise to no further trouble. On November 2, 1917, he had a contusion of the right hand.

In 1921, he was again wounded in the right arm and some months later developed a bony outgrowth in his humerus, though there is no history of any fracture of the bone. In 1921 an apparently inflammatory condition of the right arm developed which was treated by multiple incisions, and in November, 1921, a partial resection of the bony formation was carried out. He returned to work early in 1922 as a riveter, having fair movement in the right elbow.

He first came under my observation on August 17, 1922, on account of a sudden acute swelling involving the right upper arm, which was tense and swollen, with the skin red. Pain was caused by pressure on any part of the swelling. The appearance suggested a diffuse hæmatoma of the arm rather than acute infection. Treatment was by rest.

At the end of a month the swelling had disappeared, leaving faint traces of bruising. The arm was massaged and an attempt made by passive movement to improve the range of action of the elbow-joint. This treatment was followed in a few days by a recurrence of the acute swelling and pain. This settled down again in a few weeks but recurred on November 17, 1922. A Wassermann reaction taken at this time proved to be fully positive. He has undergone a course of anti-syphilitic treatment since then, and has had no further acute symptoms. The range of movement at the elbow-joint has slightly improved though the bony outgrowth remains the same.

Though it is too early to judge the final outcome, it would appear that a syphilitic myositis was the cause of the acute symptoms.

### Case of Syphilitic Osteomyelitis involving the Elbow-joint.

By C. MAX PAGE, D.S.O., F.R.C.S.

H. J., MALE, aged 26. The case is shown on account of the clinical and X-ray appearances being, in the first place, suggestive of a tuberculous lesion. The patient first noticed stiffness in the elbow in December, 1919. This

gradually became worse in spite of various treatments. When he came under my observation in August, 1922, there was a diffuse swelling in the lower-third of the right forearm. Slight oedema was present together with considerable tenderness on pressure. The elbow was held flexed to an angle of about 135°, and only slight movement in either direction from that position was possible. An X-ray photograph taken at that period showed a rarefying osteomyelitis with some periostitis of the lower 3 in. of the right humerus; slight lipping of the articular surfaces of the elbow-joint was also shown to be present. A Wassermann reaction proved to be strongly positive.

A full course of antisyphilitic treatment consisting of nine weekly doses of N.A.B. in increasing strength was carried out; mercury and potassium iodide were also administered orally.

Within a few months the clinical signs have disappeared, there is no pain or swelling and the movements at the elbow-joint are practically normal in range. X-ray examination shows slight sclerosis of the lower part of the humerus but very little alteration in its contour.

### Case of Swelling of the Face.

By DOROTHY HARE, M.D.

E. T., A WOMAN, aged 29, machinist, has for two years noticed a swelling of the lips. No history of exciting cause. She thinks that it started rather suddenly, as she remembers waking up one morning and noticing it for the first time. The swelling has gradually increased since the onset but varies in degree from time to time. She says that it is always worse when she is at work and is better on Sundays or when she is on a holiday, and that it quite disappeared during August, 1922. Since she has been under observation (three months) there has been no marked alteration; if anything a slight increase. Patient gave up work three months ago. Headache has always occurred with the swelling, which is moderately severe.

Past history: Has always been healthy except for some (?) "nervous trouble" a few years ago, for which she did not lie up.

Family history: Mother and father strong, four sisters and one brother healthy.

Examination: Healthy, and no evidence of disease elsewhere.

Local condition: Diffuse swelling of the lips with congestion. Right side more involved than left. Right side of mouth retracted and naso-labial fold deepened. Lips stiff and immobile and cannot be closed even during eating and drinking. Mucous membrane everted, dry and exfoliating. Nose slightly swollen and sometimes definitely congested. Skin otherwise normal and tissues not obviously altered in consistency, but patient says that the cheeks feel stiff. No abnormality detected in tongue, buccal mucous membrane, throat or bony parts. Nose and antrum clear.

X-ray examination of skull: No bony change visible. Pituitary fossa apparently normal. Special examinations for evidence of pituitary changes negative. Vision and fields of vision normal. Fundus normal. Sugar tolerance normal.

Wassermann reaction negative.



**Case of Mycosis Fungoides.**

(Under the care of JAMES MCCLURE, F.R.C.S.)

Shown by H. ETHELBERTA CLAREMONT, M.S., F.R.C.S.

PATIENT, a man, aged 49, has for the last fourteen months had "lumps" coming up on the skin. The "lumps" vary in size from that of a halfpenny-piece to that of the palm of the hand. They begin as smooth rounded elevations, then the surface ulcerates leaving a raw discharging area, and later they commence to heal. They disappear in course of time, their duration depending on their size and varying from a few weeks to many months. They are not painful except when they are rubbed.

Patient, a farm hand from South Wales, has been working up to his admission into hospital a week ago. Has never been abroad. Works with cattle and horses, but so far as he knows has not handled any sick animals. Has never met anyone who has had similar eruptions, nor are any of his family affected.

His general condition is good. He presents numerous flat irregular cutaneous tumours, varying from  $\frac{1}{2}$  in. to 5 in. in diameter. They are raised up to  $\frac{1}{2}$  in. from the level of the skin, their surfaces are uneven and are covered with granulations which discharge thin watery matter and bleed easily. They are freely movable and do not invade the deeper tissues, nor is there any induration round them. They are present on the chest, abdomen, arms, forearms, thighs and back, but the head is free. All the stages of their development can be observed, the earliest appearing as small rounded elevations, bluish pink in colour, quite soft, but not fluctuating; the fully developed stage as described above; and the healing stage, where epithelialization has begun, most often starting in the centre, but in some places commencing peripherally. The last stage is a well formed, mobile non-pigmented, slightly depressed scar. The skin is dry and scaly, but there are no erythematous patches. The axillary glands and the submaxillary lymphatic glands are slightly enlarged, but no others are palpable. Spleen not enlarged. Wassermann reaction negative.

Blood count: Hæmoglobin, 50 per cent.; erythrocytes, 4,430,000; leucocytes, 18,900; polymorphonuclears, 81 per cent.; lymphocytes, 10 per cent.; large mononuclears, 9 per cent.

The microscopical section exhibited has the appearance of a richly cellular granuloma, the cells varying greatly in size and shape. No giant cells visible. Active mitosis can be seen in many of the cells.

The case is shown on account of its extreme rarity, and in order to invite confirmation or refutation of, or an alternative to, the diagnosis of mycosis fungoides, and to receive suggestions for treatment.

Dr. F. PARKES WEBER agreed that this remarkable case was one of mycosis fungoides "d'emblée" (without there having been any so-called "pre-mycotic dermatosis"). He thought a trial of Röntgen-ray treatment might be made. In regard to the severity of the case, it reminded him of one which he (Dr. Weber) had described in the *British Journal of Dermatology* (London, 1918, xxx, p. 7), and which was also illustrated in Dr. J. M. H. MacLeod's book on "Diseases of the Skin."

**Case of Heart-block.**

By O. LEYTON, M.D.

MALE, aged 78. Diagnosis, complete heart-block with auricular fibrillation. In 1910 he sought advice at the London Hospital complaining of "thumping of the heart." He gave a history of syphilis forty-five years previously; had never had rheumatic fever; noticed slow pulse previous to seeking advice; always a good walker, but noticed present trouble after a long walk. Pulse (rested), 35; (exercise), increased up to 52.

Admitted to hospital, where it was found that the only drug which had any effect upon the pulse-rate was cactus and that caused a peculiar double beat.

The patient attended regularly for many years, and there was no appreciable alteration in his pulse-rate until May, 1922, when it was found to be 60. The



sign of complete heart-block, namely, pulses in the neck of three amplitudes, was absent. A cardiogram (*see figure*) was taken by Dr. Parkinson which showed that the patient had developed auricular fibrillation. The explanation of the sudden increased rapidity of the ventricular beat is a matter for discussion.

**Two Cases of Erythræmia (Vaquez Disease) treated by Röntgen-Therapy.**

By E. STOLKIND, M.D.

SINCE 1892-95, when Vaquez described his first case of erythræmia, about 250 cases have been reported in literature. Many methods of treatment have been tried without success.

*Case I.*—Patient, a man, aged 50, complained of attacks of giddiness, weakness, &c. He had always enjoyed good health; was a skilled workman. About five and a half years ago he had the first attack of vertigo, and after some months again. Since then he has these attacks at frequent intervals.

About five years ago he was in a London infirmary suffering from hæmorrhoids and loss of considerable quantities of blood, and during that time he became so weak that he could not move. In October, 1920, and later, in 1921, he was in two London hospitals. In one of them venesection was tried but without improvement. In February, 1921, he had hæmoptysis.

In December, 1921, he came to hospital with severe cyanosis of the face, nose, ears, lips, tongue, mouth, with bluish-red fauces and hands, and cyanotic clubbed fingers. Conjunctivæ injected. There were many cutaneous telangiectases on the face; the temporal arteries were tortuous. The veins were dilated. He had varicose veins in both legs. The spleen was felt, about two fingers' breadth below the costal margin, and was tender; the liver was also enlarged and palpable. Heart and aorta normal. Ophthalmoscopic examination by Dr. Harry Campbell and Mr. Lindsay Rea revealed abnormally large veins in each fundus but no cedema (venous hyperæmia or fundus polycythæmicus). The urine contained albumin. The patient denied syphilitic infection, and the Wassermann reaction was negative. Brachial blood-pressure was 140 (systolic) and 90 (diastolic) (Riva-Rocci instrument). Patient complained of occasional cramps in the legs and sometimes in one or both hands. There was often vertigo; he was easily tired, depressed, specially during the attacks of giddiness. Pain in the right mandibular joint off and on upon movements of the joint. Sometimes sensation of numbness in the legs and occasionally throughout the right side of the body. He was unable to follow his occupation.

All these symptoms and the examination of the blood (see below) show this to be a characteristic case of erythræmia (splenomegalic polycythæmia (rubra) vera). I have to thank Dr. Braxton Hicks for the blood-examination.

I began the treatment by potassium iodide, with slight subjective improvement during the first weeks only. Blood-counts on March 31, 1922: Erythrocytes, 9,500,000; hæmoglobin, 150 per cent.; colour index, 0·8; white cells, 16,400; the differential count of white cells gave: Polymorphonuclear neutrophils, 71 per cent.; eosinophils, 2 per cent.; large mononuclears, 10 per cent.; small mononuclears, 14 per cent.; basophils, 1 per cent.; transitionals, 2 per cent.

From April 24 to June 12, 1922, I tried benzol from 3 to 6 c.c. *per diem*, but without any improvement. Blood-counts on May 5: Red cells, 10,000,000; hæmoglobin, 160 per cent.; white cells, 16,400; colour index, 0·8; polymorphonuclear neutrophils, 66 per cent.; eosinophils, 4 per cent.; large mononuclears, 2 per cent.; small lymphocytes, 26 per cent.; transitionals, 2 per cent. Blood-count on May 27: Red cells, 9,650,000; hæmoglobin, 160 per cent.; colour index, 0·8; white cells, 20,400; polymorphonuclear neutrophils, 76 per cent.; eosinophils, 1 per cent.; large mononuclears, 4 per cent.; small mononuclears, 18 per cent.; transitionals, 1 per cent.

In June there was lichen planus rash over all the body (Dr. A. Eddowes). In July he complained of pain in the ileo-cæcal region; has been six weeks in the West End Hospital for Nervous Diseases. During the last six months has been treated by sodium iodide, little improvement resulting.

Blood-count October 20: Red cells, 7,700,000; hæmoglobin, 140 per cent.; white cells, 21,000; polymorphonuclear neutrophils, 81 per cent.; eosinophils, 1 per cent.; large mononuclears, 1 per cent.; small mononuclears, 17 per cent.

From November 16 to December 29, 1922, Dr. J. H. Douglas Webster has given eleven full applications of intensive Röntgen rays (38 cm. alternate gaps)

to sternum, thighs, legs and arms. A considerable improvement has resulted from the X-ray treatment.

*Case II.*—Owing to the kindness of Professor H. Guggenheimer I have had the opportunity during the past summer of examining the following case: A woman, aged 43, has been suffering at times for the last two months from bleeding from the nose and gums, headaches, tinnitus aurium, red face, rush of blood to the head. Blood-count: red cells, 8,300,000; white cells, 14,800; hæmoglobin (Sahli), 115; brachial blood-pressure, 200 (systolic), 140 (diastolic, Riva-Rocci). Traces of albumin. Spleen palpable; thyroid gland enlarged. This was a case of erythræmia, and X-ray treatment was applied.

Blood-counts before the application of X-rays: Red cells, 7,600,000; white cells, 15,100; hæmoglobin, 105 per cent. Eight days later, before the application of X-rays: Red cells, 8,200,000; hæmoglobin, 167 per cent.; colour index, 1.01; white cells, 26,000; polynuclear neutrophils, 87 per cent.; eosinophils, 1 per cent.; small lymphocytes, 6 per cent.; large lymphocytes and transitionals, 6 per cent. Immediately after the first application to the right thigh: Red cells, 8,100,000; hæmoglobin, 134 per cent.; white cells, 23,600. Four days later, before the third application of X-rays: Red cells, 6,720,000; hæmoglobin, 129 per cent.; white cells, 18,600. Directly afterwards: Red cells, 7,000,000; hæmoglobin, 126 per cent.; white cells, 18,600. Ten days later, before the fifth X-ray application: Red cells, 8,100,000; hæmoglobin, 126 per cent.; white cells, 21,600; polynuclear neutrophils, 83 per cent.; eosinophils, 3 per cent.; small lymphocytes, 10 per cent.; large lymphocytes, 3 per cent.; transitionals, 1 per cent. Directly after application of X-rays: Red cells, 8,620,000; hæmoglobin, 129 per cent.; white cells, 27,000; polymorphonuclear neutrophils, 82 per cent.; eosinophils, 4 per cent.; small lymphocytes, 10 per cent.; large lymphocytes, 3 per cent.; transitionals, 1 per cent. Before seventh X-ray application: Red cells, 7,000,000; hæmoglobin, 135 per cent.; white cells, 17,000. After the application: Red cells, 6,200,000; hæmoglobin, 140 per cent.; white cells, 20,000. Five weeks after the beginning of the X-ray treatment, but before the fourteenth application: Red cells, 6,920,000; hæmoglobin, 130 per cent.; white cells, 14,200. After the treatment: Red cells, 6,600,000; hæmoglobin, 128 per cent.; white cells, 11,400. Ten weeks after the last X-ray application: Red cells, 5,640,000; hæmoglobin, 123; leucocytes, 11,200. Seven months after the last application, by examining the patient, I have found: Red cells, 8,500,000; hæmoglobin, 115 per cent.; white cells, 10,900; colour-index, 0.7; brachial blood-pressure, 190 (systolic), 130 (diastolic). Spleen not palpable; thyroid gland enlarged; considerable subjective improvement.

*Remarks.*—Thus in the first case, that of the man, the venesections, which according to Türk, John Parkinson, Elistratov (unpublished cases, Moscow), and many others, relieve the subjective symptoms, did not give relief to this patient. In another case, that of a woman, whom I saw in 1911, there was some subjective improvement after a series of venesections.

Benzol treatment, introduced by Koranyi, Kyrallyfi, and tried by Arnstein, Barker, Beltz, McLester, Moewes, Türk, and others, had no effect on my patient. Benzol, even in large doses (3 to 6 c.c. *per diem*), had neither reduced the number of red cells and hæmoglobin, nor of white cells, and did not improve the subjective condition of this man. It is a dangerous drug! Sodium or potassium iodide alone did not reduce the count of the red and white cells, and of the hæmoglobin, and, except for a short time, did not improve the subjective symptoms of the patient. Rest in hospital for six weeks relieved the

subjective condition, but only slightly. Probably, owing to the rest, the red cell count was reduced. So far I have not tried injections of blood serum from a patient with pernicious anæmia, and Dr. Parkes Weber has been unable to find the record of such a case in the literature. I advised an iron-free and vegetarian diet. Mosse thinks that X-ray therapy of the spleen is useless. In both of the above-mentioned cases X-ray treatment applied to the bones was useful. It relieved the pain and cramps in the limbs, and generally improved the subjective condition of the male patient. He is much stronger; the limbs are not so heavy; there are no more painful cramps in the limbs; he can now easily move his head in all directions; can walk freely, and is not so shaky. But as yet he is still unable to follow his occupation. In the second case there was also a subjective improvement after application of X-rays to the bones; the count of the red cells was reduced from 8,200,000 to 6,600,000, and that of the white cells from 26,000 to 11,400. John Parkinson, Stengel, Luedin, Vaquez, Guggenheimer, Béclère, Boettner, Moench, Pendegrass, Strauss, Fraser, Seeliger, Gutzeit, Schoening, Hoegler (five cases of erythræmia treated by radium), and some others have reported cases of erythræmia treated by X-rays with satisfactory results. Of course, this kind of treatment must be controlled by repeated enumeration of the red and white cells, &c. (see the case of Brieger and Forschbach). The lapse of time will show whether the effect is durable.

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### A Simple Instrument for withdrawing Serous Effusions.<sup>1</sup>

Designed by H. O. GUNEWARDENE, M.B., B.S., and  
B. W. CANTRELL.

THIS instrument is a simple modification of the ordinary "Record" syringe, and is intended for use in the place of the more complicated apparatus employed for withdrawing pleural and other serous effusions. It differs from the "Record" syringe in that:—

(1) A side tube A is fixed to the metal mount at the bottom of the syringe to carry away the fluid which is drawn up in the ordinary way into the glass barrel of the syringe. This side tube has a tap (D) to which is attached a length of indiarubber tubing.

<sup>1</sup> For the loan of the block illustrating the instrument, the designers are indebted to Messrs. Allen and Hanbury.

(2) The ordinary needle is replaced by a cannula, which can be screwed on to the barrel of the syringe.

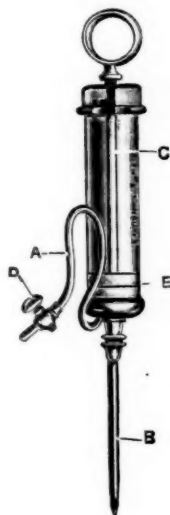
(3) A trocar (C) is screwed on to bottom of plunger (E).

All the parts of the instrument are detachable, and trocars and cannulae of various sizes can be obtained.

The instrument is used like a "Record" syringe when employed for exploration. To aspirate, the fluid to be removed is drawn into the glass barrel; the tap is then opened and syphonic action is started by pushing down the plunger half an inch or so. The plunger is then steadied and the fluid will continue to flow into the glass barrel and thence through the side tube (A) into any receptacle.

The advantages of the instrument are:—

(1) It is simple, easy to carry about and to sterilize. Cannot get out of order, and has no indiarubber connexions to perish.



(2) Ordinary syringe needles can be attached, and it can then be used as a simple syringe.

(3) Exploration and removal of fluid can be done at one sitting, and with the least amount of discomfort to the patient.

(4) Thick fluids, for example, pus, can be pumped away, the trocar fixed to the plunger, automatically closing the opening of the cannula and hindering the return of fluid into the serous cavity.

(5) The instrument can be used for withdrawing pus from, and subsequently irrigating or injecting any fluid into, serous cavities, joint cavities, or abscesses.

(6) It can be used for introducing gas into the pleural cavity after or during the process of withdrawing fluid.

(7) The withdrawal of fluid can be done steadily and slowly, and the operator is able to see exactly what is occurring during the operation.

(8) The disadvantages of most aspirators now in use are eliminated.

The instrument can be obtained from Messrs. Allen and Hanbury.



## Clinical Section.

President—Sir WILLIAM HALE-WHITE, K.B.E., M.D.

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### Case of Bilateral Calculous Pyonephrosis Ten Years after Double Nephro-lithotomy.

By PHILIP TURNER, M.S.

THIS patient was shown at a meeting of the Clinical Section on May 2, 1913,<sup>1</sup> when the following notes of the case were submitted: E. G., aged 42, was admitted on January 18, 1913, for pain in both loins and pyuria. The trouble was first noticed eighteen years before, when she passed two small stones, and pus was noticed in the urine. Four years after this she was admitted to the St. Albans Hospital, where another stone was passed. She then remained well for four years, when another calculus the size of a date-stone was passed. The patient then continued in good health until August, 1912, and since then she had had pain, worse on the left side, also pyuria and hæmaturia. On admission both kidneys were greatly enlarged, extending down into the iliac fossæ. On the left side a very large calculus could be distinctly felt, while on palpating the right kidney numerous stones could be detected, and a sensation like crepitus could be readily obtained. The urine was acid in reaction, and contained much pus, blood and albumin. She was very thin, pale, and cachectic. A radiographic examination showed numerous shadows in the right kidney, and a large dark area in the left. On January 23, the left kidney was exposed, and a large calculus, weighing when dried 4 oz., and several smaller ones were removed; two large abscesses containing foul pus were also opened and drained. She stood the operation well, and no serious symptoms followed. Four weeks later the right kidney was exposed, and seventeen calculi and a number of small ones were removed, their total weight being 4 oz. The pelvis of the kidney was distended with foul turbid urine. The patient made good progress, and was discharged on March 13. Though still of a bad colour she has put on flesh, and is much better and stronger. The urine is now acid in reaction, contains no blood, and only a trace of albumin. The wounds are firmly healed, and the kidneys appear to be about normal in size.

I had not seen or heard of the patient since 1914, until she appeared at my out-patients two weeks ago. She had been free from pain, and felt perfectly well until five weeks ago, when a small swelling appeared in the middle of the scar on the right side. This burst, pus came away, and a little discharge still persists. No urine has been noticed to escape from the sinus while she



has been under observation. Both kidneys are palpable, but they are not tender, and are not enlarged. The lower pole of each is above the level of the umbilicus. In the middle of the right scar is a small sinus, along which a probe can be passed as far as the muscles.

X-ray examination shows a large opacity in the lower pole of the right kidney, and in addition there are one or two vague shadows, probably areas of gritty deposit, to be seen in both kidneys.

Examination of the urine: Specific gravity 1011, reaction acid; microscopical examination of the deposit shows a few pus cells but no casts; there is a trace of albumin. Bacteriological examination showed a pure growth of *Bacillus coli*. A blood-urea examination showed 0.44 gr. per 1,000 c.c., which is slightly more than normal. The urine concentration test showed a considerable deficiency in concentration. Before the examination the urea was 1.23 per cent.; after one hour, 0.94 per cent.; after two hours, 1.04 per cent.; and after three hours, 1.25 per cent.

### Case of Exophthalmos probably caused by Non-suppurative Cavernous Sinus Thrombosis.

By F. PARKES WEBER, M.D.

THE patient, M. T., is a married Englishwoman, aged 28, with slight exophthalmos of the right eye, and more of the left eye. She can see well with the right eye, but is blind in the left eye. There is much chronic firm swelling of the left lower eyelid (especially subconjunctival tissue), with ectropion and chronic conjunctival discharge (see figure, p. 42). She has a presystolic apical murmur from old mitral stenosis, though she has no knowledge of ever having had rheumatic fever. The history is that about June, 1921, she had "quinsy," which lasted three weeks, and was followed by "swelling" of both eyes. The right eye became much the worse of the two, but in December, 1921, the swelling in the right eye began to subside, whilst that in the left eye increased. I first saw her about that time (December, 1921), when the striking feature of the case was the reddish oedematous swelling of both eyelids of both eyes, together with protrusion of both eyes, more marked on the right side. Dr. R. Gruber, who saw the case first, reported that there was right-sided optic neuritis. By Röntgen-ray examination of the skull nothing abnormal could be found to account for the ocular condition, and the nose and throat had likewise been examined with negative result. By examination of the blood and viscera, chloroma and leukaemia could be excluded, and there was no evidence of any tumour in the thorax or abdomen. There was no enlargement of any superficial lymphatic glands. The blood-serum (December, 1921), gave a completely negative Wassermann reaction for syphilis. The urine was free from albumin and sugar. The patient was in about the fourth month of pregnancy, that is to say, she had become pregnant in August, 1921, after the eyes had already commenced to "swell."

In February, 1922, she seemed well, except for the eye condition, which, however, had become decidedly worse, at all events on the left side. There was "choked optic disc" on both sides, more marked in the left eye than in the right. There was much exophthalmos on both sides, with great injection of the deeper vessels over the sclerotics. On the left side there was considerable conjunctival chemosis about the inner canthus. By fresh Röntgen-ray

examination the pituitary fossa appeared to be not enlarged, and there was nothing to suggest any disease in the sphenoidal or frontal sinuses. There was apparently no fever. Against a diagnosis of Graves' disease I would especially mention the absence of thyroid enlargement and of Graefe's sign, &c., the presence of only slight tachycardia (connected doubtless with her pregnancy and anxiety), the vascular injection of the eyeballs, the swelling of the eyelids, the conjunctival chemosis and the optic neuritis ("choked discs"). Mr. G. J. Jenkins, who kindly examined the patient, suggested that the eye condition



To show the appearance of the eyes in March, 1923. From a photograph kindly taken by Dr. Marshall.

might be due to non-suppurative thrombosis of the cavernous sinuses, and expressed himself against any operation. He had seen a somewhat similar case, in which the signs gradually subsided, the improvement commencing on the side which was at first the worse of the two.

In March, 1922, the protrusion of the left eye had increased, and there was more conjunctival discharge on that side, whereas there was slight improvement on the right side. From March 22 to May 6, 1922, the patient was in the hospital, and was treated by rest and local cleanliness and protection for the

eyes. There was no fever. At the commencement of June, 1922, the patient gave birth to a healthy child (probably a little over term) in a maternity hospital. (She had already two children, both of them living and healthy, one born in 1919, the other in 1920.)

Since then the protrusion of both eyes has diminished, but she cannot see with the left eye. In the right eye ophthalmoscopic examination still shows slight optic neuritis; the vision is  $\frac{6}{12}$ .

In regard to causation, the only probable suggestion seems to be a non-suppurative (at first progressive) cavernous sinus thrombosis, commencing after the attack of "quinsy," and rendered worse by pregnancy, which latter alone is an occasional cause of vascular thrombosis. Canalization of the thrombus has doubtless taken place, but the circulation in the orbits has not yet quite returned to the normal, at all events on the left side. In all probability a spongy structure, like a venous angioma, has replaced the original free cavernous sinus channel.

Temporary swelling of the pituitary gland, sometimes (like temporary thyroid swelling) connected with pregnancy or menstrual periods, may, as Dr. Kinnier Wilson points out, give rise to the rare symptom, "hemianopia fugax," but could not give rise to the features of the present case. We have no proof that there was suppurative sphenoidal sinus disease (a possible cause of local vascular thrombosis), and a tumour at the base of the skull can be almost excluded, though of course it must be admitted that tumours are well-known causes of venous thrombosis.

It might be objected that if canalization of the thrombus had taken place, the proptosis on both sides ought to have completely disappeared. The proptosis has, indeed, become much less on both sides, especially on the right side, but canalization of large blood-vessels does not mean *restitutio ad integrum*. It is a gradual process by which new channels may be formed, not only in the thrombosed vessels themselves, but, I believe, also in the remains of the inflammatory material (if there is any) around them. This process substitutes a sponge-like circulation for the original open channel, so that the resistance to the passage of blood remains always greater than it was before the thrombosis took place. It may, moreover, be surmised that some of the newly-formed blood-vessels are prone to become obliterated by fresh thrombosis.

### Hemiplegia occurring in Pregnant Woman at Full Term ; Sudden Onset accompanied by Transient Albuminuria ; Cæsarean Section ; Gradual Recovery.

By FRANK COOK, F.R.C.S.

PATIENT, aged 41. Seven previous confinements without noteworthy complications. Recent confinement due January 10, 1923. She was knocked down by a ladder on December 10, 1922, and slightly concussed (?) Sent to hospital on January 1, 1923, as a case of "albuminuria of pregnancy"; but no albumin found in the urine (except a trace on January 8, 1923) until immediately after her attack on January 11, 1923. Breech presentation corrected by external version on January 2, 1923. No other abnormality found. Blood-pressure 130.

January 11, 1923: No albuminuria in the morning. Between 6 and 7 p.m. she had eight or nine "fits," after an entirely uneventful afternoon, followed by unconsciousness with incontinence, &c. 8 p.m.: Blood-pressure 120. Now no evidence of foetal vitality. Plasma bicarbonate 0'0153 molar conc. (normal, 0'035). Urine: "Loaded" with albumin (small catheter specimen only), pH 5'0; no sugar, no blood; granular casts.

January 12, 1923: No further fits. Semiconscious. Incontinent. No albuminuria.

January 13, 1923: Right-sided hemiplegia now definite, with aphasia. Patient conscious. No albuminuria.

January 16, 1923: Blood-pressure 145. Plasma bicarbonate 0'0314. Urine, pH 5'1. No albumin. Wassermann negative. No sign of labour.

January 17, 1923: Cæsarean section (and sterilization by resection of tubes). General anæsthesia. The foetus had apparently been dead for several days—there had been no sign of foetal life since the first attack.

March 3, 1923: Discharged from hospital. Walks well. Movements of arm and hand fairly good. Speaks fairly well. Difficulty in reading. Occasional headaches.

The chief points of interest in this case are the exact diagnosis of the cerebral lesion, the association of the attack with temporary albuminuria and with the death of the foetus *in utero*, and the evidence of acidosis. Although apparently far removed from a typical eclampsia, it is always possible that such a case might afford some clue towards the solving of that most fascinating pathological problem.

### Case of Graves' (Parry-Graves-Basedow) Disease in a Woman, aged 69, without Goitre.

By E. STOLKIND, M.D.

CASES of Graves' disease (hyperthyroidism) in old persons are very rare. Harry Campbell has seen only one other case in a patient as old as this. Charcot reported such a case in a patient, aged 54. Eppinger reported two such cases in patients aged 54 and 56 respectively. Barker has seen a case in a woman aged over 60.

A woman, aged 69, complained of general weakness, wasting, nervousness, &c. The onset began about eight years ago, when, according to her own history, she became ill from worry: She lived in buildings of one-room flats; two noisy people lived next door. She could not rest at night. Sometimes after this she fell out of her chair, but did not lose consciousness; later on she had only two similar attacks. Dr. Harry Campbell then found very marked exophthalmos, wasting, tremor, tachycardia, &c., but the thyroid gland was not enlarged. She suffered also from palpitation, diarrhoea, perspiration, nervousness, &c. After treatment she improved very much: she had no more palpitation and sweating; exophthalmos and tremor became very little visible. This state of health lasted for about six years. During the last two years had two relapses with marked exophthalmos, tachycardia, trembling of the legs, hands, and tongue. During the relapses the cardinal symptoms of Graves' disease were more marked than now. The pulse is now 100 to 126, regular. The heart is slightly enlarged to the left, and the radioscopia confirms this diagnosis. The patient is very thin and became emaciated; weighs 6 st.

3 lb.; eight years ago weight was 8 st. All her teeth were extracted on the ill advice of her doctor. The patient is tremulous and nervous, though not so much as eight years ago. Graefe's symptom is now negative, while before it was positive. There is a brown pigmentation on the skin. A year ago I treated her for urticaria. She complains now and then of dryness in the mouth and roughness in the throat. There has never been any sign of an enlarged thyroid gland or of a substernal goitre. Under X-ray examination Dr. G. Fildes has found that the chest is clear; no evidence of any retrosternal mass; line of trachea normal in both views, antero-posterior and lateral. All the patient's other organs are normal in correspondence with her age. This case of Graves' disease without goitre is in favour of the hypothesis of the influence of thyroid toxæmia in the pathogenesis of hyperthyroidism. The prognosis in such cases is not bad. The treatment should be symptomatic.

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## Cases of Adiposis Dolorosa (Dercum's Disease).

By E. STOLKIND, M.D.

*Case I.*—Patient, a very corpulent woman, aged 35, complains of pains in different parts of the body, of feeling of great tenderness upon palpation, especially the breasts, legs and arms, of gradually progressing general weakness, nervousness, &c. The patient comes of a nervous family with tendency to obesity: both parents were stout: the eldest brother was very remarkably so. From childhood she could not see well with the right eye. She was a well-developed, rather thin girl until she married at the age of 20. She soon became pregnant, and began to grow stout. After the first child was born, her weight was 13 st. 5 lb. During the next five years she gradually put on more fat, and nine years ago her weight was 17 st. 9 lb. Her husband has had syphilis. She is the mother of four children, and there was also one stillborn child and three miscarriages. The eldest daughter is bald (complete alopecia). In spite of the obesity the patient was very strong and could move and lift very heavy things; this she was obliged to do every day. She could walk long distances, was very energetic, had a good memory. She suffered from pains, which were diagnosed as "rheumatoid." During the last three years she became gradually weaker and weaker; it is now difficult for her to walk, or to lift anything, as she becomes soon tired. She is suffering from spontaneous pains in the limbs and other parts of the body; the pains in the front of the legs are specially troublesome, mostly in the day time. Her arms are very heavy, "like lead." She complains of frequent and severe headaches and pruritus of the head. She is now very sensitive to cold and is often shivering. No perspiration (anhidrosis). She does not perspire even upon application of hot-air baths. She is becoming more and more irritable, and every trifling matter excites her; she is sometimes depressed; gradually she loses her energy, and does not take the same interest in things as before. Her character is less determined. The memory has become worse and is "dull." She is

losing her hair. She is now very adipose, weighs 15 st. 9 lb.—i.e., less than nine years ago—but the face, neck and extremities are very little affected. For the last three years, and especially during the last fourteen months, the breasts have become progressively bigger and heavier; they are monstrously enlarged, pendulous, very weighty and very tender. The abdomen and the trunk are distinctly adipose; the skin is normal. There are two lipomas on the pectoralis major muscles. All parts of the body are very tender upon being touched, especially the legs, breasts and the lipomas. Heart, lungs, &c., normal. Patient is very intelligent. Thyroid, up to 5 gr. per day, was given for two years without any improvement. (I do not know whether the thyroid she received was really thyroid gland; the President (Sir William Hale-White) has mentioned a case in which no thyroid was found in tablets marked "thyroid gland.")

General reflexes are normal. No albumin and sugar. Wassermann reaction negative (only taken once). Upon X-ray examination Dr. G. Fildes found that the sella turcica appeared to be much diminished in size, an appearance which suggests hypopituitarism.

The following case under my observation is less typical.

*Case II.*—Patient, a female, aged 46, complaining of palpitation, attacks of pain in the left side of the chest, headaches, general weakness, pain in the legs, tenderness of some adipose deposits, &c. She is stout, and some years ago was stouter still. Weight 12 st. 10 lb. Pendulous adipose deposits of upper arms; adipose apron-like abdomen and thighs. Marked tenderness of fatty deposits of thighs, legs, especially of left leg, and upper arms. No lipomas. There is muscular weakness; hitherto she was much stronger. Hysteria present; is becoming more irritable and quarrelsome; her character is changed: hypochondriacal tendency; often depressed.

Is suffering from aortic regurgitation and aortitis (luetic?). Mobile right kidney. Hyperidrosis. The two cardinal symptoms, obesity, tenderness of the adipose deposits upon manipulation, and pains, strongly support my opinion that this is a case of Dercum's disease. I am not certain about the other symptoms—except the disease of the circulatory system—being the consequences of this polyglandular disease.

*Remarks.*—The first is a typical case of *adiposis dolorosa*—Dercum's disease: Obesity, tenderness, pain—spontaneous and upon manipulation of the adipose deposits, gradually progressive asthenia, psychic changes, secretory symptoms (anidrosis), &c. The great diminution in size of the sella turcica, and thus of the pituitary gland, shows that in my case also the pituitary body is diseased. Changes in the hypophysis in cases of *adiposis dolorosa* were found at autopsies by Burr (glioma), by Dercum and McCarthy (adeno-carcinoma), by Guillain and Alquier (hypophysis doubled in size with changes suggesting an alveolar carcinoma), by Price (two cases with changes suggesting carcinoma), and by some others. In many cases changes in the thyroid gland, ovaries, &c., were also found at autopsy. The freedom of the face from fat and myxœdema, the tenderness and pains—spontaneous, as well as provoked by manipulation of the skin and fat deposits, the lipomas, &c.—all distinguish this case from hypothyroidism, as well as from *dystrophia adiposo-genitalis*.

I have ordered massage, electricity, diet and pluriglandular treatment (pituitary, thyroid and ovarium), as Dercum's disease is caused by changes of the endocrine glands.

I have also observed atypical cases of *adiposis dolorosa*, i.e., cases in which



not all the cardinal symptoms were present. In three cases there are obesity, tenderness and pains upon pressure (mother, aged 62, and her two daughters aged 29 and 25). The mother suffers from general obesity, arteriosclerosis, interstitial nephritis, anæmia, neurasthenia, &c. Diffuse adipose deposits, especially on the chest, abdomen, trunk and thighs. Touching the arms, chest and abdomen causes an outery of pain. She complains of fatigue, weakness, occasional attacks of pain in the chest, radiating to the left arm. There are also anidrosis and alopecia. The daughter aged 29 has adipose deposits on the shoulders, abdomen and thighs, which are tender and painful upon manipulation. There are symptoms of neurasthenia. The other sister has adipose deposits on the chest, abdomen, trunk and thighs, which are painful and tender upon pressure. She is an anæmic and hysterical woman.

I am unable to agree with Lyon and others, in their description of atypical cases of adiposis dolorosa in patients without obesity.

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## Cases of Post-encephalitic Paralysis Agitans.

By E. STOLKIND, M.D.

Case I.—Male, aged 20. Sister died from pulmonary tuberculosis. Parents healthy. Was a healthy and intelligent boy. At the end of 1919, at the age of 18, when preparing for his examination as a senior clerk in the Civil Service, he became ill: fever, rhinitis, conjunctivitis, general weakness, &c. The doctor diagnosed influenza. During the illness he had a heavy rhinolaryngeal breathing (like snoring), slept badly and was restless at night. Was in bed for about ten days, and was very weak when he got up. The illness left him a little "shaky" and more excitable than before. The peculiar breathing with an open mouth lasted for a few months after recovery. He was absent from the office for about three weeks, and was very much worried about his examination, in which he failed: this was in January, 1920, i.e., about four to six weeks after his illness. For some months after his illness he was restless at night and drowsy by day, and complained of pain in the left arm. He was studying for the next examination, which he passed in July, 1920. He was found by the doctor to be healthy and suitable for the post of permanent official.

Meantime he became gradually more nervous and "shaky." He was working in the office until October, 1921, when he had to be suddenly discharged owing to conditions of his health. He could not concentrate his mind, especially on figures. He was breathing through the mouth. For ten months was an out-patient at a hospital, where he had been recommended to have his tonsils and adenoids removed. The operation was performed, but without any improvement. The mouth is still open.

Gradually he became worse, and when he came to the hospital I found all the following symptoms of Parkinson's disease present, namely: Expressionless face, open mouth, but without dribbling of saliva, well marked rhythmical



tremor of both arms, legs and tongue, typical gait, trunk stooping forwards; all the voluntary movements of the trunk and limbs are stiff and slow. He is very weak, very apathetic; not able to work.

After thyroid treatment he has become brighter, takes more interest in life (for instance he now reads about and discusses wireless telegraphy and the telephone), his speech is no longer slow, he asks for more tablets. His legs, however, are still weak, and the tremor is the same. During the last few weeks he has been taking polyglandular tablets, hyoscine hydrobromide, and has been given massage.

*Case II.*—A patient, a female, aged 23, was generally healthy, and engaged to be married. In May, 1920, after tonsillectomy she went for a holiday to Devonshire, where she soon became ill; she had severe pains and a rash all over the body, &c. She was there ten days altogether, was able to come to London, and complained of severe pain all over the body, fever, general weakness, became in the same night unconscious and delirious. For a whole month she was in this state, being delirious specially at night. Gradually she began to recover, to be able to walk and to understand things; her memory improved. The diagnosis was encephalitis lethargica. Although she is a tall girl, her weight is only 6 st. 11 lb.; very pale; no headache nor pains. She walks with her head bent forward; facies is expressionless, mask-like; all the voluntary movements of the trunk and limbs are slow and stiff; she moves slowly, as if she were made of wood. There is tremor in the hands, legs, and tongue. The hands are in a typical position as in cases of paralysis agitans; the same holds good for her gait. All the other organs are normal. She does not take interest in anything, though she reads a little; mostly she is sitting like a "wax-figure." All reflexes normal. She cannot work much and soon becomes tired. Speech low and slow. Her fiancé thinks that she is not the same girl whom he wanted to marry, and wants to get rid of her. It is a characteristic case of post-encephalitic paralysis agitans.

*Case III.*—Patient, a female, aged 44. Home duties. Had seven children and three miscarriages. In March, 1920, she became ill; fever, headaches, pain in the extremities, rhinitis, lassitude, &c. Her doctor diagnosed influenza. She was in bed for two weeks; slept badly; could understand everything. When she recovered, she began to work as hard as usual. After some weeks (months?) the relatives noted that she stooped. Gradually she became worse and weaker, without power to hold anything in the hands, even a cup of tea. For the last nine months her state has been especially serious: Her arms and hands are becoming weaker; she is stooping more and more, the head falls forward, the chin rests on the chest; progressive trembling in her legs and hands. The movements in all the joints are becoming more difficult; she cannot raise her arms properly. It seems as if her joints are spastically contracted, the left arm especially. She complains of aching in the left arm on and off every day, the pain being very severe sometimes. Has noises in the head; suffers every day from severe headaches: she feels as if someone were hammering her head. There is the typical Parkinsonian mask-like face. She is not the same woman she was formerly. She is mentally defective. Sometimes she starts talking foolishly. There is nearly complete loss of memory; she forgets what she has done, what she has said, where she has put things, what she wanted; she will go twenty times for the same thing and forget each time what she wanted. She has no appetite; eats only when forced to do so. Apathy for everything.

*Case IV.*—Strong, stout girl, aged 22, coming of a healthy family; three

brothers and five sisters—all well. Has had no serious illness hitherto; used to be very intelligent.

In May, 1920, patient felt "bilious" quite suddenly and fell asleep. This state of lethargy lasted for some days. For six months she was unable to do anything, not even to speak; she was lying in bed, and had everything done for her. She gradually improved until she became "perfectly fit." About eight months later she had a relapse, getting worse again. She has never had diplopia, paralysis, or eye trouble. Menstruation ceased for six months at the commencement of her illness. When she came to the hospital she presented a typical picture of paralysis agitans. There was complete "mental lethargy." Very slow and very low speech; characteristic posture and gait; right foot dragged more than left; tremor of the limbs and tongue, increased on voluntary efforts; Parkinsonian mask; stiffness of the body with slowness of all movements, &c. Later she was admitted to the hospital, where she improved a little: she could feed herself, could walk little better, and her gait was less "log-like." She lost 7 lb. in weight in eight weeks, while she remained in hospital. I saw the patient the last time on March 5, 1923; she was lying like a log, and was unable to speak; sometimes it is possible to understand her by the movements of her lips. When she wants anything, she lifts her hand, and if not observed, then incontinence follows. She is now very emaciated; the right hand is continuously trembling, and this is more marked when she is trying to do something. There is talipes equinus.

I propose to publish in another paper details of the other two cases of post-encephalitic paralysis agitans in children aged 10 and 14. Both of them present a typical picture of the Parkinsonian syndrome. The girl, aged 14, is intelligent, quiet, very weak, only able to walk a few steps at a time and that not every day; she suffers from continuous dribbling of saliva, and for the last weeks from polyuria and polydipsia. The boy, aged 10, is able to walk when he likes. His mental state is not the same as before: he is very impulsive, irritable, often restless, very obstinate, and he often screams.

*Remarks.*—The cases shown exhibit varying grades of post-encephalitic paralysis agitans in children and adults. In two of these cases the man, aged 20, and the woman, aged 44, encephalitis lethargica was overlooked, and the illness was diagnosed as influenza. Every patient presents a typical picture of paralysis agitans with the characteristic posture and gait, the Parkinsonian mask (expressionless face), tremor of the hands, legs and tongue, slow involuntary movements of the trunk and extremities, the trunk bent forwards, and slow and peculiar speech. Spastic resistance is marked in the voluntary muscles. In one case, that of the woman, aged 22, there is loss of power of speech. In the same case relapse of the Parkinsonian syndrome occurred, about eight months after recovery. Now she is lying like a log. In one case nearly complete loss of memory is noticed. None of the patients are the same men and women as they were before the onset of the encephalitis lethargica. In one case, as stated, the fiancé does not want now to marry the girl, to whom he was engaged for a long time. In another case the husband does not know what to do with the wife who is now helpless. In one of the cases all the teeth were extracted, of course to the great disadvantage of the patient. I have under my observation another case of paralysis agitans in whom all of the patient's teeth were extracted upon the advice of a well-known physician, and the patient is now worse. These cases show that the prognosis is not a good one and that at any rate it cannot be made accurately.

**Case of Auricular Fibrillation ; Reversion to Normal Rhythm under Administration of Quinidine.**

By B. T. PARSONS-SMITH, M.D.

A. A., MALE, aged 35, by occupation a clerk. Recurring tonsillitis in childhood; rheumatic fever twice (when aged 17 and again when 20). Deficient effort tolerance after second attack renders patient incapable of work other than that of a sedentary type. First attended hospital, July, 1917, complaining of giddiness, palpitation, dyspnoea, flatulence, and pain in the precordial area; diagnosis, mitral disease with ? adherent pericardium; condition remained moderately stationary except for an attack of influenza in 1918, which aggravated the symptoms and was associated with an obstinate cough and blood-stained expectoration. Slight recurrence of rheumatism in the summer of 1922, with marked general debility and frequent attacks of palpitation; onset of auricular fibrillation during July, 1922.

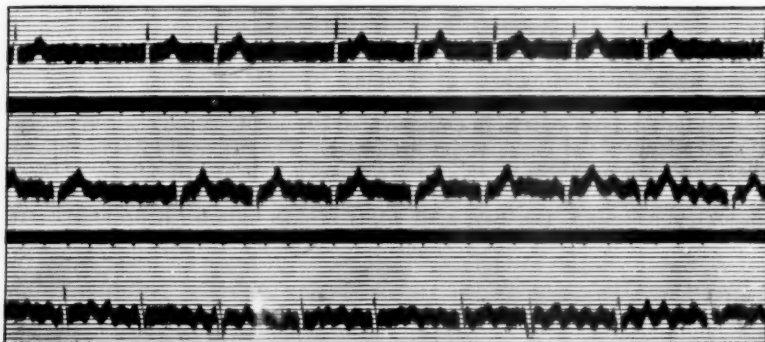


FIG. 1.

Treatment with tr. digitalis, 10 minims three times daily, controlled the ventricular rate, but patient invariably complained of palpitation, heart-flutter, flatulence, indigestion, &c., and in addition there was usually a radial pulse deficit, which varied between 12 to 20 beats per minute.

Patient was admitted to hospital on November 13, 1922, his condition then being one of mitral disease with auricular fibrillation; heart not enlarged; ventricular rate, 100 per minute; no jugular stasis; no visceral engorgement; radial pulse, 88 per minute; blood-pressure (maximum) 130 mm.; exercise tolerance poor. The preliminary treatment comprised complete rest in bed with digitalis for eight days, electrocardiograms then showing auricular fibrillation with an auricular oscillation rate of 420 and a ventricular rate of 92 (fig. 1).

On the ninth day patient was given two small doses (3 gr.) of quinidine sulphate in order to test for a possible idiosyncrasy to the drug. On the tenth day patient was ordered quinidine sulphate 5 gr., three times daily; rapid reduction in the auricular rate was observed; on the eleventh day the rate was

*Clinical Section*

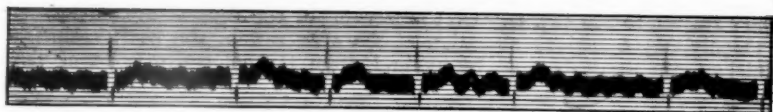


FIG. 2.

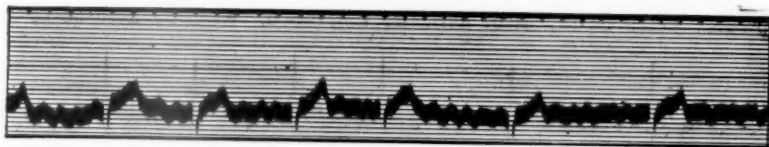


FIG. 3.

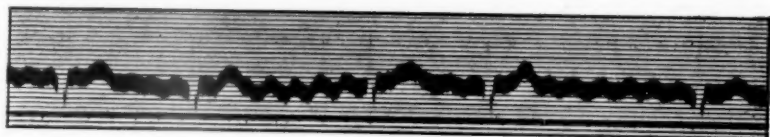


FIG. 4.

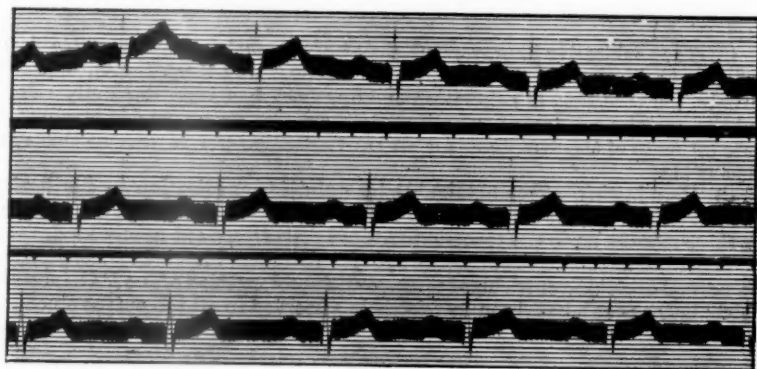


FIG. 5.

noted to be 360 (fig. 2), on the twelfth day it had fallen to 330 (fig. 3), and on the sixteenth to 250 (fig. 4). On the morning of the seventeenth day patient suddenly felt a curious sensation at the apex region, the heart seemed to quicken, the beats became heavy and, from the subjective point of view, perfectly regular.

On examination it was evident that the rhythm had reverted to the normal, and an electrocardiogram (fig. 5) proved confirmatory. Small doses (5 gr.) of quinidine were prescribed during the subsequent week, after which the drug was stopped entirely, mainly on account of persistent headache.

The case presents many features of clinical interest:—

(1) A demonstration of the power of quinidine to abort the excitation process which characterizes auricular fibrillation. (A daily dose of 15 gr.

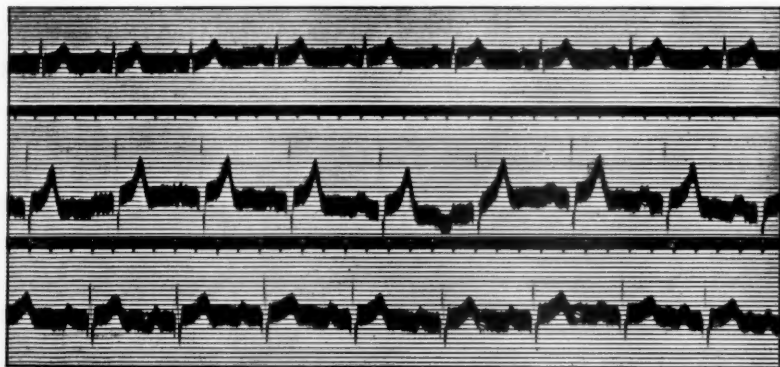


FIG. 6.

produced the desired effect in the above-mentioned case after seven days' medication.)

(2) That subjective improvement may be a sequel to the reversion from auricular fibrillation; in the present case the fluttering sensation disappeared entirely, the threshold of palpitation and dyspnoea has been distinctly raised, and the exercise tolerance has improved; these phenomena enable an approximate estimate of the symptoms for which fibrillation *per se* was in this case responsible.

(3) The lasting effect obtainable by quinidine medication in fibrillation of the auricle; in the present case the sinus rhythm has remained dominant for three months since the reversion without drug treatment of any kind.

The most recent electrocardiogram (fig. 6) taken March 2, 1923, displays the normal auriculo-ventricular sequence with prolongation of the P-R interval, which latter event has been persistently observed in all the curves since the reversion.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Two Cases of Ringworm of the Nails in Sisters.

By H. C. SEMON, M.D.

THE elder sister, aged 37, contracted the disease six years ago, and it has affected symmetrically the middle, ring and little fingers of both hands. During this time the nails have been shed spontaneously, and in the case of the little finger of the right hand twice. Microscopic examination in potash has revealed a segmented mycelial growth, from which it is hoped a culture will be secured. The toe-nails are normal.

The younger sister gives a history of six months' infection, and states that she thinks she contracted it after using her sister's manicure set. In her case the middle and little finger of the right hand, and the little finger only of the left hand, have been attacked.

The source of infection in the elder sister's case cannot be traced.

It will be remembered that recently I showed a case of ringworm of all the nails of the hands: a report of this case has been published in the *Proceedings*<sup>1</sup> and in the *British Journal of Dermatology*.<sup>2</sup> I now pass round a culture on standard acid glucose agar, which has been isolated and prepared by Dr. W. Broughton-Alcock, chief pathologist to the Ministry of Pensions. During the development of the fungus the medium has changed colour from its normal yellowish tinge to a decided red, and the rugose surface of the white culture has also assumed a pinkish tinge. Dr. Alcock and Dr. Castellani are of opinion that the case must be regarded as one of infection by the *Epidermophyton rubrum*. Members will remember that it was decided to remove all the nails and treat the nail bed subsequently (as suggested by Dr. MacLeod's experience), with weak mercury ointment. This was done, and the patient was removed to a convalescent home, with strict injunctions to keep his nails covered in finger stalls. It appears, however, that the directions were not followed, as he was in the habit of using the roller towel in the annexe of the ward. Ten days after the patient's admission, two ward maids developed tinea circinata of the shoulder and sacral regions respectively, and a third ward maid developed ringworm of the nails of two fingers of the left hand seven weeks after his admission. The disease has recurred in all the nails in the original case.

<sup>1</sup> *Proceedings*, 1922, xv (Sect. Derm.), p. 31.

<sup>2</sup> *Brit. Journ. Derm.*, June, 1922, p. 208.



It would therefore seem that ringworm of the nails must be regarded as a more infectious condition than is generally described in text-books, and I think it my duty to publish the unfortunate facts of my original case, in the hope that catastrophes of this nature may be avoided in the future.

#### DISCUSSION.

Dr. PERNET said that in 1901<sup>1</sup> he showed two cases of undetected tinea unguium in sisters. One patient, aged 41, had had the complaint for twenty years. Scrapings showed *Trichophyton megalosporon endothrix* mycelia, and he confirmed this by a culture. In the other patient, aged 44, the nails had been affected for twenty to thirty years. The same fungus was demonstrated in scrapings, but culture failed. The former patient did well on Harrison's treatment. Both cases and the culture, &c., were shown at the old Dermatological Society of London. In the case of a girl, aged 15, with ringworm of the scalp and nails, Dr. Pernet had obtained cultures of *Trichophyton megalosporon endothrix* from both hair and nails. That from stumps was of a delicate, pale lilac-pink.<sup>2</sup>

Dr. W. K. SIBLEY considered that the proper treatment for these cases was ionization by zinc or copper salts, the whole of the affected finger nail being submerged in a beaker containing the solution to be applied, together with the positive electrode, the negative or inert one being applied to the opposite hand. He had never avulsed a nail for this disease.

### Case of Lichen Planus Annulatus, with Atrophy and a Herald Patch.

By GEORGE PERNET, M.D.

PATIENT, a housewife, aged 46. Duration of rash, two months. It is distributed about the trunk and limbs, presenting a number of ringed areas; some have coalesced. The centres of most of the larger ones are depressed and atrophic in appearance. Smaller ones have a very fine border. Here and there characteristic discrete lichen planus papules are present. The mouth is not affected. A small ring,  $\frac{1}{2}$  in. across, with a delicate border, in the centre of the upper part of the chest over the manubrium sterni, appeared first, and was present a month before the rash became generalized. This may be compared with the herald patch of pityriasis rosea, and also with the primary patch of psoriasis, in which latter condition a single patch only may be present for some time before generalization occurs. There was some pruritus when the patient was first seen, due to a great extent to sulphur ointment, which had been applied on the advice of a chemist. The patient is improving and doing well on mist. hydrag. biniodidi.

### Case of Epidermolysis Bullosa.

By HALDIN DAVIS, F.R.C.S.

THE patient, a young man, aged 26, is an example of a mild degree of the condition. His mother noticed it in his early infancy soon after he had been vaccinated, and it has persisted ever since. Any slight trauma, such as that

<sup>1</sup> Pernet, *Brit. Journ. Derm.*, xiii, 1901, p. 268, and xiv, 1902, p. 16.

<sup>2</sup> Pernet, *Brit. Journ. Derm.*, xviii, 1906, p. 252.

produced by lying on a crumpled portion of the sheet of his bed or by a slight knock on the limbs, causes the appearance of a blister. Notwithstanding this serious disability the patient for some years persisted in playing football and in consequence his shins have suffered very severely, and still are frequently the seat of bullæ of large size. He also succeeded in doing two years' service in the R.A.M.C. during the war, most of which he spent on a hospital ship, where he had opportunities of dressing his legs and concealing his condition from those in higher authority. Ultimately however it was discovered, and he was discharged from the Service, but he is now on the staff of the Ministry of Pensions. Unlike most cases of this disease his teeth are good and his nails, although affected, are not very badly formed. Since taking quinine regularly he appears to have shown some improvement.

### A Note on Molluscum Contagiosum.

By J. JACKSON CLARKE, M.B.

(Introduced by Dr. G. PERNET.)

FOR the material on which my observations have been made, I am indebted to many dermatologists, beginning in 1894 with Mr. (now Sir) Malcolm Morris, who placed in my hands for pathological examination some typical specimens of lesions freshly excised. Since that now remote date I have lost no opportunity of observing the behaviour of the characteristic molluscum corpuscles when placed in surroundings that allow it to be shown whether they are living things.

The object of this paper is to induce others who, having command of material, have also time and inclination to repeat and extend, or to correct, my observations.

The necessary apparatus need not occupy more than a square foot of space in a consulting room, and cannot but be pleasing to the eye, two pairs of Petri dishes being all that is required besides the microscope, slides and covers, mounted needles, wire loop, &c., which already find their places.

The patient's skin should be cleansed with absolute alcohol, no strong antiseptic being used. Water<sup>1</sup> should be taken after the tap has been allowed to run a few seconds, in order to remove traces of copper, and boiled, as also should be all watch-glasses, Petri dishes, slides, &c.

A watch-glass is placed in the lower of each pair of dishes and some water (not enough to float or cover the watch-glass) is poured into the lower dish (see fig. 1). One dish is left covered by its upper half; in the watch-glass contained in the other is placed a little heap of excised lesions cut open from the surface downwards; or of the white wax-like material that can be obtained from ripe lesions by lateral pressure. For small portions a cupped slide raised above water-level by two other slides being placed under it may replace the watch-glass. A few drops of water, but not enough to cover the heap, are now added, and the upper dish is inverted over the lower.

<sup>1</sup> I have had no opportunity of making cultures with rain-water, which might suit better than tap-water. Other variations of the culture medium will suggest themselves as worth trial. I have found that streaming occurs with normal saline, and the addition to the latter of a trace of bicarbonate of soda favours subdivision of the bodies into sub-equal segments.

The culture must be kept at room temperature, because incubation favours the growth of bacteria, which, when over-abundant, kill the specific parasites. Occasionally a culture apparently free from bacteria is obtained, but usually bacteria are present; in moderate number they do not prevent vital changes from occurring.

It is important to make clear drawings of objects at the time they appear under the microscope, and it assists the eye if the main features of each stationary part are traced by means of a drawing eye-piece or other similar optical instrument.

*Enumeration of Vital Changes.*—Some vital changes which I have already observed are shown in fig. 2. They are (1) streaming of protoplasm with reproduction by budding; (2) formation of a supporting framework and of protective capsules; (3) formation of bird's-eye bodies; (4) vacuolation with oscillation of granules; (5) formation of active flagellate or spirillar bodies.

One of my latest observations was made in November, 1919, on material kindly provided by Dr. G. Pernet, Physician in charge of the Department for Diseases of the Skin at the West London Hospital, and the house-surgeon,

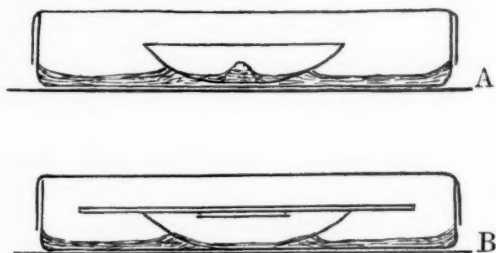


FIG. 1.—A, the material to be cultivated is heaped in the middle of a watch-glass; and water, but not enough to cover it, has been added drop by drop; B, after teasing in a drop of water on a slide a particle or drop of the material in "A" is covered and examined microscopically, then inverted over a watch-glass containing water and put between Petri dishes for re-examination later.

Dr. Barker. I have also to thank Dr. Beverland and Mr. H. K. Shaw, resident medical officers at the Hampstead General Hospital, for helping me with the preparations. The specimens were placed in covered Petri dishes, some in tap-water as described above, others on spent tea-leaves in water.

All the preparations gave positive results. I was able to demonstrate the movement on the third, fourth, fourteenth, and fifteenth days to seven different trained observers. In these demonstrations the movement was shown under a magnification of 500 diameters with a dry objective. The oil-immersion lens when used for preparations simply mounted in water has the drawback of dragging on the cover-glass and causing movements in the objects beneath it, but it must be used to study certain features.

The preparations from Dr. Pernet's case I last examined on the thirty-fifth day after making the cultures. On this occasion streaming was present in a few only of the corpuscles, and the rest were obviously degenerating and becoming stained by the colouring matter of tea-leaves on which they were placed. So long as they remain in full vitality they do not absorb the colouring matter from the tea-leaves.

(1) *Streaming*.—The reality of the phenomenon being thus established a few details may be added. Fig. 2 (a) shows the aspect of a corpuscle just before streaming begins; its texture becomes more granular: this is succeeded by a disappearance of granules from either part of, as at (b), or throughout a corpuscle, as at (c), (e), and (f). The fresh corpuscle often presents indications of being segmented, and a regular segmentation is sometimes seen in water cultures; in one such I found the rotary motion affected one only of the segments, fig. 2 (d). There are no separate granules to be seen in the moving substance, which must be composed of extremely minute elements,<sup>1</sup> but large granules are sometimes seen on the surface of the flowing part of a corpuscle; such granules I have observed to be carried round one segment of bodies such as those shown in fig. 2 (c). Thus it appears to me that the phenomenon is a real streaming of protoplasm; at any rate it is conspicuous and quite different in aspect from effects produced by bacteria, motile or stationary.

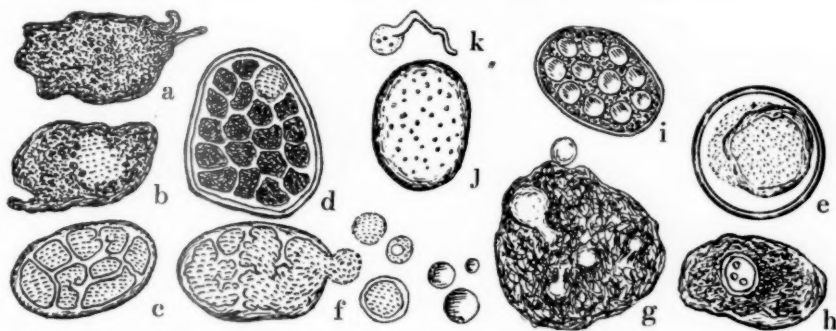


FIG. 2.—a, Molluscum body unchanged except that the cortex is more definite and the granules are larger; b, a clear area has formed and in it the protoplasm is circulating; c, an internal framework continuous with the cortex has formed and the whole of the protoplasm is circulating; d, a capsule has formed and within it the protoplasm is subdivided into segments, in only one of which the protoplasm is circulating; e, a spherical capsule has formed, the protoplasm is circulating and is in part protruded beyond the cortex; f, the whole protoplasm is circulating and a small protrusion is present, and there are three separate subdivisions with circulating protoplasm, one vacuolated, another with a clear outer zone, and, to the right, three subdivisions as they appear when they come to rest, clear and refracting; g, a body in which five subdivisions are seen in process of formation by budding; h, a body in which a nucleus-like structure has formed; i, a body with vacuolated protoplasm, the intervening granules in Brownian movement; j, a body with violent agitation of protoplasm; k, flagellated body with agitated granules at its expanded end. d, f, and g,  $\times 800$ ; the rest  $\times 500$ .

*Budding*.—From some of the bodies with streaming protoplasm, part of this can be seen to protrude, as in fig. 2 (f), and in the neighbourhood are smaller spheres with streaming: in some of the latter there is a bright outer zone with no streaming. Some of the small streaming spheres have a vacuole. All three kinds when they are observed to come to rest assume a uniform refracting appearance and have a green tint. Although I have not seen the actual separation of one of these smaller bodies, after watching protrusions such as are shown in fig. 2 (f) and (g), I have no doubt that they are formed by budding from the large bodies.

<sup>1</sup> The uniform and all but homogeneous appearance of the streaming substance is quite in harmony with the filtrability of the fresh corpuscles after they have been ground up with sand and suspended in saline solution.

(2) *Doubly contoured capsules* are rare in fresh material, but many develop in water cultures, fig. 2 (c). They seem to be formed as a defence against bacteria; I have seen many with bacteria on the outside of the capsule but never inside.

(3) *Internal Framework*.—Irregular processes extending inwards may be formed apparently in the spaces between the segments of a corpuscle. They are sometimes regular and anastomose to form a net, fig. 2 (c), or the framework<sup>1</sup> may be scanty as at (f).

(4) *Nucleus-like Bodies*.—These are not present in the fresh corpuscle. Granules appear in an area where a nucleus or bird's-eye body is to be formed. Oscillating motion may be seen among the granules as the nucleus appears in a clear space bounded by a membrane. This stage represents, in my opinion, a low state of vitality of the molluscum body.

(5) *Vacuoles*.—These I have seen once as sketched in fig. 2 (i). The granules between the vacuoles were in a state of oscillation or Brownian movement. I have seen a similar appearance in a sclerotium of that much-studied and interesting protist *Badhamia utricularis* after it had been twelve hours in water.

(6) *Flagellate Bodies*.—This striking feature I have only once observed in a culture on the fourth day. The material was from abundant lesions, some of which I heaped up in the hollow of one of the cupped slides for hanging-drop preparations, and placed in a moist chamber as shown in fig. 1. The result I described in the *Centralblatt für Bakteriologie*, 1895, i, p. 245.

The description, slightly condensed, runs:—

"The most remarkable appearance consists in the presence of a great number of actively-moving flagellate bodies. They have a roundish head of the size of a red blood corpuscle, and a single powerful flagellum, and under a one-twelfth immersion lens were easily seen and unmistakable; many passed across the microscopic field and then escaped from sight. Many of the molluscum bodies were unchanged; of others, but a thin shell remained; still others had apparently undergone a liquefaction in their central part, and in this area were numerous highly-refracting oscillating granules."

I may add that in what I called the heads of the motile bodies (see fig. 2 (k)), there were oscillating particles quite like those present in those of the molluscum bodies I described as liquefied in the middle. The room in which the culture was made, in March, would be at a temperature not much above freezing at night, and for some hours each day somewhat above blood-heat, the preparation being placed on a chimney-piece above a bright fire.

The "heads" may be only residual, the flagellum becoming independent as a spirochæte or spirillum. I regard this state of reproduction as the acme of vitality in the cultivated parasite, the streaming state with budding coming next.

Answer is sure to be desired to one question: "Suppose we do as you suggest and see changes such as you describe, and even agree that they prove that the molluscum body is a parasite; in what biological text-book can we find an account of other parasites of the same genus?"

<sup>1</sup> In material given me by Dr. Ernest Dore last year I tested for cellulose some of the bodies in which a framework had formed by adding a few drops of a mixture of iodine solution (one part) to sulphuric acid (two parts). Parts of the framework changed to a greenish blue colour, as much reaction as I have ever found in the fungal cellulose of moulds, mildews, &c. With the same reagent the molluscum bodies changed to a deep purple which lasted a few days, and the epithelial cells became pale yellow and swelled to oval form. Some *Synchytrium taraxaci* I treated in the same way and found its capsules were unaffected; the sporangial contents became dark green.

Taxonomy has no group in which we can find place for this parasite for which I have proposed the name *Plassomyxa contagiosa*.

Natural history still has some blank pages : one of these I am now asking you to help to fill.

Many have tried to identify the molluscum body with the familiar genus *Coccidium*. That attempt has failed.

Only by purely objective study can we know the characteristics of an organism, and having ascertained them with minute care we must, if necessary, make a new group in classification. The group to which the causal parasite of molluscum belongs I have named the Plassomyxineæ.

Scattered in cryptogamic botany are papers on species of *Synchytrium*, and in protozoology, accounts of Haplosporidia and Rhizomastigina; the parasite of molluscum has affinities with these and allied groups. They are all protists, and the related literature is part of protistology.

The unnatural divorce of cryptogamic botany from protozoology accounts for some of the delay there has been in recognizing the molluscum body as a parasite.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case of Leishmaniasis of the Skin.

Shown by H. C. SEMON, M.D., for J. B. CHRISTOPHERSON,  
C.B.E., M.D.

THE patient is a military nursing sister, single (aged 28), and the disease was first manifested in December, 1920, as an indolent raised pustule on the left cheek, three months after a fly bite, received while she was nursing at Baghdad. Leishman-Donovan bodies were demonstrated at Netley at the end of February, 1921. By the middle of June the small ulcer had healed, under application of antimony ointment, boric ointment and X-rays, and she was discharged apparently cured, although "a small raised lump, neither yellow nor red" was still present. In November, five months later, the "raised spot" seemed to grow larger, and since then other spots have come out, one by one."

She was first seen by Dr. Christopherson on June 9. There were about twenty-five little vesicular infiltrations, of a yellowish colour, quite soft on palpation (fig. 1). The skin was not ulcerated, a scar marked the site of the original "oriental" sore. There was no complaint of pain and the lymphatic glands did not show any enlargement. No typical Leishman-Donovan bodies were found, but two atypical, probably degenerated forms were demonstrated, in a scraping, by Dr. Christopherson.

Treatment by intravenous injections of sodium antimony tartrate was begun on June 12, and by June 28, when  $8\frac{3}{4}$  gr. had been injected, the nodules appeared to have become flatter, more solid, and scaly on the surface. At the present time they are almost on a level with the surrounding skin, and practically indistinguishable from lupus vulgaris, which was the diagnosis made by myself when I was first consulted, some three weeks ago. Eighteen grains in all of the sodium antimony tartrate have been given up to the present date, and the treatment will be continued so long as improvement is maintained.

A photograph of a very similar case (under Dr. Christopherson's care), also in a nursing sister, who has had the disease on the cheek for a year (fig. 2), supports the view that we are here dealing with a new and hitherto undescribed form of dermal leishmaniasis, resembling lupus vulgaris in its clinical appearances. This patient has refused intravenous sodium tartrate treatment, but her condition is clearing up very gradually in the country under applications of methylene blue paint.

[July 20, 1922.]



Leishmaniasis of the skin, lupoid form. Nursing sisters, both originally contracted "oriental sores" at Baghdad. (Leishman-Donovan bodies present.) After a period of (presumed) cure, grouped, nodular, cystic, non-ulcerated lesions appeared, the nodules containing semi-fluid, apple-jelly substance.



FIG. 1.—These nodules appeared about the original site five months after the primary Baghdad sore was "cured." The scar of the original sore is visible. The above appearance is eight months after the nodules were first noticed. After the administration of 18 gr. of sodium antimony tartrate by intravenous injection (five weeks), the lesions were level with the surrounding skin and appeared as dull yellow infiltrations of the skin. ? Degenerated Leishman-Donovan bodies found.



FIG. 2.—In this case the original sore occurred on the right arm. The nodules appeared on the right cheek some months after the arm-sore was "cured." The above photograph was taken one and half years after they first appeared on the face. The case was not treated by intravenous injections of sodium antimony tartrate but seemed to be slowly improving with an alkaline solution of methylene blue. Leishman-Donovan bodies were not found.

## DISCUSSION.

Dr. A. WHITFIELD agreed with the diagnosis. He said that a little more than a year ago a case occurring in an officer had been shown by Dr. Graham Little. The Leishman sore had been excised, and was followed by an eruption indistinguishable from lupus. At that time he (Dr. Whitfield) thought the clinical appearances were deceptive, and that it was really relapsing leishmaniasis. He had talked with those who had had a large experience of Leishman sores, and they said these sores did sometimes relapse. This was the third case which had been seen at the Section, and what they had to decide was, whether it was likely that the cases constituted a form of leishmaniasis with which members were so far not familiar. Or was it likely that in the three cases named the curious coincidence had occurred of leishmaniasis setting up tuberculous lupus? The former idea seemed to be so much the more likely, that he thought it must be considered that on rare occasions this kind of tropical sore did develop.

Dr. W. DYSON said that when the slow development of lupus was taken into account he did not think that, without examination, one could exclude the likelihood of lupus having been engrafted on to a sore which had been caused by Leishman's infection. Clinically, this case was indistinguishable from a case of lupus. Lupus could often be seen developing on so-called septic sores of a chronic and stubborn character.

Dr. GRAHAM LITTLE said that in the case to which Dr. Whitfield had referred there was never any convincing evidence of leishmaniasis; the Leishman-Donovan bodies were never demonstrated in the skin in that instance. He agreed with Dr. Dyson that these patients might subsequently develop tuberculosis. He had shown several times the case of a young girl who had both syphilis and tubercle; she first had a gumma, and on the gumma there developed a typical tuberculous sore, which did not heal. One must be very sure of a diagnosis before accepting a new classification. It was remarkable that such a thing should not have been noticed before. Sir William Willcox, whose patient this was, and who had had a large experience in Mesopotamia, was convinced that the condition was not leishmaniasis.

Dr. J. H. SEQUEIRA commented on the fact that the two cases described were in women, in whom tuberculous lupus was commoner than in men. He did not regard the present case as complete; a portion of the lesion should be removed and examined microscopically.

Dr. ARTHUR POWELL said he had been twelve years in Assam, the home of kala-azar, but had never seen any eruption of this kind associated with it. During a further period of twenty years he had seen many cases of Delhi boil arising in India and Irak but nothing like Dr. Christopherson's case. It seemed to him a case of lupus arising on the scar of the Delhi sore.

Dr. J. M. H. MACLEOD considered that the lesion was tubercle which had probably been grafted on the original disease.

Dr. H. G. ADAMSON (President) said that without knowledge of the history one would not hesitate to diagnose lupus vulgaris in this case. The cheek was a very common site for lupus. He was in the habit of teaching that the disease was common in that situation because of the infection from the nostril along the course of the lymphatics. He did not think there was conclusive evidence that this case was leishmaniasis. He had seen cases of Delhi boil which in the later stages had presented the appearance of lupus nodules; but none so apparently typical of lupus as the present case and he thought it important that a further attempt should be made definitely to prove it to be a leishmaniasis if it were so.

Dr. SEMON (in reply) said that the case as it stood was admittedly incomplete, and would be submitted to further investigations, but he was of opinion that considerable improvement had taken place already since the treatment had begun. If the patient

should refuse to have a biopsy done, a complement-deviation test would be carried out for tubercle. If that should prove negative, it would be against the diagnosis of lupus vulgaris, and therefore in favour of leishmaniasis. It was hoped that the case would be shown again at the October meeting.

## Two Cases of Chronic Erythema of the Legs.

By H. MACCORMAC, C.B.E., M.D.

THESE two patients present closely similar appearances, viz., chronic erythema of the legs, and as this condition appears to have been more commonly seen of recent years, they have been brought to-day for demonstration. It will be remembered that Dr. Dore<sup>1</sup> recently exhibited a similar case, and that some discussion took place as to the nature and cause of the eruption. The first of the two patients is a woman, aged 42. She gives a history of tubercular glands, and has recently suffered from acute pleurisy, possibly of a tubercular nature. About two years ago she developed a patch of erythema on the leg in the region of the ankle. There is a remarkable freedom from subjective sensations except some aching. It is noted that the erythema increases and becomes more obvious when the leg is in the dependent position, and it lessens when the leg is kept elevated. Thus it almost entirely disappeared when the patient was confined to bed during the recent attack of pleurisy. The second patient is a young woman, aged 29. There is no history or evidence of tuberculosis in any form, and her general health is good. In her case the erythema, which is limited to the lower aspect of both legs, began some three years ago. She complains of burning sensations in the affected area, but otherwise the eruption does not cause any discomfort.

In these two cases the objective condition is similar. It is more superficial than the type met with in some forms of Bazin's disease, and it does not appear to be directly related to alterations in temperature, as it remains unchanged throughout the year. A number of examples of this form of erythema have recently come under my observation in hospital, and it would seem now to be relatively common.

### DISCUSSION.

Dr. H. W. BARBER said that he had recently met with a case of this kind—that of a nurse who was rendered incapable of doing her work owing to œdema of her legs, accompanied by erythema. In his experience the clotting-time of the blood was notably prolonged in these cases, and this suggested the probability of some abnormality in calcium metabolism.

Dr. SEMON said that he had at present under his care a similar case in a young woman, in whom the chief trouble was constipation. Since the constipation had been relieved the skin condition had improved. Galvanization had been useful in that case. He regarded it as a sort of vascular paralysis, due to intestinal toxæmia.

## Case of Parapsoriasis.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a lady, aged 45. She has lesions over a large part of the body, which commenced to appear five years ago; they have increased in size, in the characteristic way of parapsoriasis, and I have little doubt that the

<sup>1</sup> *Proceedings*, 1922, xv (Sect. Derm.), p. 43.

condition is parapsoriasis *en plaque*. The largest lesion is round the left breast, and it is entirely devoid of infiltration: that is the most important criterion in the decision between mycosis patches, which are like this, and parapsoriasis.

### Case for Diagnosis (? Leukæmia Cutis).

By W. KNOWSLEY SIBLEY, M.D.

W. N., A SHOP assistant, aged 41, was sent to me by Dr. Harkness, on March 21 of this year. He was more or less covered by a macular eruption which had been present for a month. There was nothing to note in his family history; he had served in the war and been twice wounded, and never had any illnesses before. The rash first appeared on the forehead, and at that time was very abundant on the face, arms, legs and chest. There was slight irritation at night. The rash had been diagnosed as a secondary syphilitic eruption, and certainly in many respects was very suggestive of that disease. There were, however, no lesions of the mucous membranes, enlarged glands, or any other symptoms. The Wassermann reaction was found negative, and again negative after a provocative injection. After a short time, the eruption in many parts became papular, and this was especially noticeable about the scalp and forehead.

April 12: Differential blood count showed moderate eosinophilia, but no other abnormality. Total leucocyte count normal, but total red blood corpuscle count showed slight polycythæmic condition. No obvious abnormality in size, shape or hæmoglobin content of red cells.

Blood examination: Total leucocytes, 11,120 per cubic millimetre; total red cells, 6,880,000 per cubic millimetre. Differential count: Polymorphonuclear cells, 62 per cent.; small lymphocytes, 24 per cent.; large lymphocytes, 2 per cent.; eosinophils, 5 per cent.; basophils, *nil*; large hyaline cells, 4 per cent.; transitional cells, 3 per cent.

After a few weeks, the rash disappeared from the whole body for a time, and then reappeared on the forehead, and gradually over the whole face and scalp, and more or less over the rest of the body. The eruption was now distinctly papular, and in places nodular, often of a peculiar purplish coloration, especially about the forehead and tip of the nose, and many of the lesions were painful to the touch. Papules then appeared on the limbs, especially about the legs; these were also of a purplish colour. There was also at this time an extensive eruption inside the mouth, affecting especially the hard and soft palate, and the patient complained of sore throat. Lymphatic glands, especially about neck and groins, were generally much enlarged, and the supratrochlear glands stood out very prominently. The glands were isolated, hard and firm, with no signs of breaking down. From time to time there was a rise of temperature to 101° and 102° F., lasting three or four days, accompanied by a pulse of 120, again becoming normal. Liver considerably enlarged, some four fingers' breadth below costal arch; spleen slightly so. Neither albumin nor sugar in the urine. The patient was obviously ill, and also complained of rheumatic pains about the legs, and of progressive weakness and loss of flesh. Recent Wassermann and sigma tests again both negative.

Differential leucocyte count, June 9: Polymorphonuclear cells, 51 per cent.; large lymphocytes, 5 per cent.; small lymphocytes, 33 per cent.; eosinophils, 5 per cent.; basophils, *nil*; large hyaline cells, 5 per cent.; transitional cells, 1 per cent.

Clinically the case rather resembles rapidly progressive Hodgkin's disease (pseudo-leukæmia), with very extensive adenitis, enlargement of spleen and notably so of the liver ; attacks of intermittent fever and pains in the limbs. On the other hand itching has been almost absent, and this is usually a prominent symptom of Hodgkin's disease.



Case for diagnosis (? leukemia cutis). Appearance on July 6, 1922.

#### MICROSCOPICAL REPORT ON THE CASE.

(1) *Section of Large Nodule (July 6, 1922) from Scalp.*—(a) Epidermis very much stretched and thinned to a layer of two, three or four cells in thickness. In one or two places where the subjacent infiltration reaches it, the infiltration is passing through the epidermis and the initial stage of ulceration is shown. (b) Corium and hypoderm: This is entirely filled by an infiltration which reaches up to the epidermis in places and extends down into the subcutaneous fat. It completely surrounds the sebaceous glands, sweat-ducts, lymphatics and capillaries, passes between the alveoli of the sweat-glands and the lobules of the subcutaneous fatty tissue. The glands,

capillaries, &c., appear normal and not involved in any way by the surrounding process, but there is almost a complete absence of collagen bundles. The characteristic cell of the infiltrated area is a polygonal cell with a rather large pale irregularly-shaped nucleus showing a very distinct nuclear membrane, a nucleolus, but otherwise little chromatin. There are a few small round cells and fibroblasts also present. Mitotic figures are very rare and the method of infiltration of the subjacent fat alone is suggestive of a diffuse sarcomatosis. The general histological picture is one suggestive of mycosis fungoides in the pre-mycotic stage, but examination of lymphatic glands should be made before an exact diagnosis can be arrived at.

(2) *Section of Small Nodule (July 6, 1922) from Forehead.*—Shows similar appearances to that of large nodule, but there is more infiltration of epidermis with the characteristic cells.

(3) *Previous section (March 21, 1922) from lesion on right chest* showed similar appearances to above.

July 19, 1922: Wassermann and sigma reactions both negative

W. ARTHUR YOUNG, M.B.

#### DISCUSSION.

Dr. T. C. GILCHRIST (U.S.A.) said the case reminded him of one of myeloid leukæmia which Dr. Kettering worked up at the Johns Hopkins Hospital. When the patient first came to the hospital the case looked like typical leprosy, but no bacilli were found. As in the case now shown the eruption disappeared. On the section of the first eruption no one would commit himself—not even Dr. Welch. But it was very definite now, and it was probably myeloid leukæmia.

Dr. W. DYSON said that his colleague, Dr. Savatard, had a case with very similar lesions on the face, in which he diagnosed leukæmia cutis. Apparently the blood in that case was normal, but large numbers of lymphocytes were found in the tumours themselves.

Dr. WILFRID FOX remarked that nearly all cases of leukæmia of the skin had characteristic slaty-blue plum-coloured nodules, which this patient did not show.

Dr. WHITFIELD thought Members should consider whether this was an atypical case of Kaposi's disease. The sections were obviously inflammatory, not true sarcoma, and they showed none of the lymphoid follicle architecture which was seen in true leukæmia of the skin, but rather blocks of pigment, which he considered homogeneous in origin. In the later stages of some cases of Kaposi's disease there was glandular enlargement.

Dr. T. C. GILCHRIST (U.S.A.) said this section did not suggest Kaposi's disease to him. A case of myeloid leukæmia showed nothing abnormal in the blood, and the other lesions could not be recognized. In a myeloid case the cells were of mixed shape, as they had not had time to form a true cell. In the case of which he had been specially thinking, there was the typical blood-picture before the patient died.

Dr. MACLEOD said that in Hodgkin's disease the eruption frequently consisted of itchy papules. He had had two such cases which turned out to be Hodgkin's disease, one being diagnosed at the examination, the other by biopsy of a gland. The eruption in the first of these cases looked like scabies.

Dr. SIBLEY (in reply) said the question of the case being one of multiple hæmorrhagic sarcoma had occurred to him, especially considering the condition inside the mouth, which, seen by the naked eye, was very similar to that of a case he had shown before the Section some time ago of multiple hæmorrhagic sarcoma with lesions of the buccal mucosa.<sup>1</sup> The great rapidity of the course of this case, however, was against that idea.

*Note.*—The patient died a week later.—W. K. S.

<sup>1</sup> *Proceedings*, 1920, xiii (Sect. Derm.), pp. 127-132.

## Case for Diagnosis.

By H. C. SEMON, M.D.

PATIENT, a woman, aged 61, came to the hospital six weeks ago with this eruption, which began in the summer of 1919. She says it came on after a walk, when she became very hot, and the eruption has continued ever since. A week later it appeared on the face. It was never itchy, but there is some slight soreness. Her history throws no light on the ætiology and her Wassermann reaction is negative. She refuses to have a biopsy done.

## DISCUSSION.

Dr. H. G. ADAMSON (President) thought this was an example of what had been called "the adult type of urticaria pigmentosa." Cases had been shown by Dr. Douglas Heath, Dr. Dore, and by himself; and recently Dr. Wallace Beatty had published notes and a photograph of a case in the *British Journal of Dermatology*.<sup>1</sup> These cases were of interest from the point of view as to whether they were really an adult type of urticaria pigmentosa or a disease *sui generis*, as Dr. Whitfield had at one time suggested. The lesions were small pigmentary macules, which occurred upon the trunk and limbs; they were only slightly urticarial on friction and it seemed not quite certain whether they always contained abundant mast-cells. They were often mistaken, at first, for secondary syphilis.

Dr. WHITFIELD said that in a case he had seen the eruption was on the arms only. All the instances of it he had seen had been in women. He did a biopsy, and there were some mast-cells, but no such aggregation of them as was found in cases occurring in children.

Dr. GRAHAM LITTLE said that on one occasion two cases of urticaria pigmentosa in men were shown at the Section; the disease did not seem to have a sex differentiation. He did not think the present case was urticaria pigmentosa; the lesions seemed to be too homogeneous. In adults they were entirely flat. If urticaria in adults was an entity, it differed from that in children in some important respects.

Dr. SIBLEY said these lesions occurred most abundantly on the trunk; seldom, if ever, on the limbs alone.

## Case of Acne Agminata.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a man, aged 35, has had the eruption for the past four months. There are numerous small waxy yellowish nodules closely aggregated, in the way Crocker had emphasized, above the eyebrow, the eyelids, the sulci below nose and cheek and on the cheeks. There is little or no necrosis at present, but there are some pitted scars of former lesions which have necrosed. There are no other symptoms of tuberculosis and there is no tuberculous history.

<sup>1</sup> *Brit. Journ. Derm.*, 1921, xxxiii, p. 151.



### Case of Acne Agminata.

Shown by H. MACCORMAC, C.B.E., M.D., for Captain BRUCE,  
R.A.M.C.

THIS man, a soldier, apparently in robust health, reported sick on June 7, with a rash on the face. The eruption consists of numerous tiny red-brown nodules distributed mainly on the forehead and cheeks. These nodules undergo a necrosis, leaving behind small scars. The condition appears to correspond with Crocker's acne agminata.

### Case of Syphilis in a Man.

By WILFRID FOX, M.D.

AT first, this appeared to be a simple straightforward case of syphilis, but one or two points made its nature doubtful. He ran the risk of infection on January 20, he had a primary sore on the penis on February 12, which would be about the right date. On February 22 he was examined in Dublin, and the *Treponema pallidum* found on the sore. The Wassermann reaction was weakly positive. The diagnosis was made of a recent infection from that date. He was given four injections of novarsenobillon, and half-way through that course the Wassermann reaction was strongly positive. Each time after an injection a rash appeared and became worse, therefore the injections were stopped. He came to me on July 3, and he then had a lichenoid rash on the arms, some of the papules were isolated, some had run together in big solid plaques. He had a diffuse syphiloma of the lips; and a typical late glossitis and leucoplakia of the tongue. In my opinion, therefore, this is an old infection, showing a chancre redux, and not a recent contamination.

### Desquamative Erythema associated with Arthritic Changes.

By Sir JAMES GALLOWAY, K.B.E., M.D.

(Shown by M. G. HANNAY, M.D.)

(I) M. G. HANNAY, M.D.

PATIENT, a boy, aged 15. Father alcoholic. Mother healthy. Patient youngest of three children, the other two physically healthy, but one is in a mental home. He has always lived in this country and was apparently quite well until two years ago, when he had an attack of what was called influenza, lasting one week. Almost immediately afterwards it was noticed that the fingers of both hands were flexed and stiff, and that the skin of the hands and fronts of legs was reddened and in parts scaly. There was apparently no pain, but considerable itching. Desquamation followed with improvement in the skin condition, but it seems doubtful if it entirely disappeared. The fingers remained flexed and stiff. In March, 1922, there was a recurrence of the skin

trouble involving the hands, forearms, knees, back and ? face. At the same time the elbows became fixed in a flexed position, but there was little if any pain. He was then admitted to a hospital in Richmond where he remained about a month, having ointments rubbed into the skin, the condition apparently improving considerably. After leaving the hospital there was an immediate relapse and he was admitted to Charing Cross Hospital.

Condition on examination: Intelligence below normal. Nutrition poor. He is emaciated and looks ill. Glands in neck, axillæ and groins, enlarged. Mouth dry, teeth coated; oral sepsis present. Heart normal. No enlargement or disease of any abdominal organ discovered. Urine: Specific gravity 1028, acid, no sugar; trace of albumin. Tendency to profuse perspiration in somewhat localized areas, especially over epigastrium. Lungs: Clinically no evidence of disease. X-ray report (May 3, 1922): Definite increase in hilum shadows, more marked on right side, and increase in peribronchial striation, suggestive of chronic infection of bronchial glands, and some fibrosis of lungs. Sella turcica, X-ray report (May 10, 1922): Normal in appearance. Blood (May 16, 1922): Rouleau formation normal. Red blood cells, 4,600,000; hæmoglobin, 80 per cent.; colour index, 0·8; white blood cells, 7,800. Differential count of white blood cells: Polymorphs, 51 per cent., small lymphocytes, 41 per cent., large lymphocytes, 2 per cent., eosinophils, 3·5 per cent., basophils, 0·5 per cent., large hyaline, 2 per cent. No changes in stained red cells. Joints: Joints of elbows, wrists, and hands, more or less stiff and flexed. There appears to be some slight relative bony enlargement at some joints, especially those of the index fingers, metacarpo-phalangeal joint of ring and little fingers, and knee-joints; but there is no increase of free fluid, no crepitus, no thickening of mucous membrane, no lipping nor osteophytic out-growths. Very slight degree of anterior subluxation at wrist-joints. X-ray report: Wrists and hands: Bones somewhat atrophied, and terminal phalanges distinctly so. Some increase in translucency of carpus and lower ends of radius and ulna. Marked atrophy of shaft of radius. Elbows: No marked bony changes. Wassermann reaction negative. Serum: no agglutination with members of enteric group of organisms. Fæces: No evidence of the presence of abnormal micro-organisms. Skin, face: symmetrical areas of desquamative erythema affecting chiefly inner portion of lower lids, side of nose, and slightly so inner part of upper lids, sides of cheeks, front of ears and forehead. These areas are flat, uniform, with fairly well-defined margins. Desquamation manifested in thin soft flakes, easily removed. No evidence of special follicular involvement, and no induration. Some degree of atrophy, and tendency to ectropion. Eyebrows and lashes unaffected. Skin of body as a whole moist, thin and supple. Certain well defined areas, chiefly, but not entirely over points of pressure, involved in scaly, erythematous condition, which in some parts, e.g., the back, hands, and forearms, covers almost the whole surface as a sheet. In other parts, such as the abdomen, there are only a few small scattered lesions, varying from the size of a large pin's head to that of a shilling, mostly circular, but sometimes running together and forming irregular figures.

The lesions themselves are uniform, erythematous, in parts atrophic. There is constant desquamation in large thin flakes, which fall off very easily. In parts subject to constant friction of the clothes, such as the anterior aspect of the knees, the scales are so constantly rubbed off, that smooth, dry, red, shiny surfaces are left. On scratching with a blunt curette, on parts not subject to such friction, no "tache de bougie" appearance is produced; large,

delicate, thin, somewhat moist, flakes are easily removed, leaving a smooth, shiny, slightly moist surface, with minute hæmorrhagic points. The lesions show scarcely any infiltration, but they tend on the one hand to fine atrophic scarring, and on the other, to continuous parakeratotic desquamation. There is no congestion of the individual papillary loops in the skin papillæ. This point seems to differentiate these lesions distinctly from the psoriasis group. The skin of the forearm and backs of hands is paler and smoother. Instead of being supple it is tense and cannot be picked up, thus showing a sclerodermic change. The patient complains of slight itching. The nails of the fingers are deformed and incurved, and show fine parallel ridges extending over about one third of the nail. The rest of the nail is fairly smooth, and no pits are seen. The toe-nails are affected to a less extent. There is a very marked generalized pityriasis of the scalp.

#### (II) Sir JAMES GALLOWAY, K.B.E., M.D.

This patient is an example of the association of psoriasiform dermatitis, with atrophy of the skin resembling certain stages of sclerodermia, and deformity and fixation of joints resembling certain forms of arthritis. The question raised by such cases is whether it is right to describe this malady as psoriasis or sclerodermia or arthritis? It seems probable that the condition is not true psoriasis, is not true sclerodermia, and not necessarily a primary arthritis. Several cases similar to that of the patient have been under my notice during the past twelve months. In certain of these the arthritic element is the most prominent, in others the sclerodermic element is the most marked, whilst in others the desquamative dermatitis is the overshadowing condition. In the present case the stiffness of the joints of the extremities and the inflammation of the skin seemed to occur simultaneously. The atrophy of the skin might be coincident or secondary to the dermatitis. It is a question whether true arthritis exists or whether the changes in the joints may be due to a slowly progressive inflammation of bone.

The suggestion made is that these cases are due to the same or similar general infective processes. In certain cases the scaly skin disease is most prominent; in others the sclerodermic change, whilst in still others the bony changes with deformities are most pronounced. The nature of the infection is not yet identified. It does not seem probable that the degenerative processes are associated with endocrine deficiency.

#### DISCUSSION.

Dr. H. G. ADAMSON (President) said he thought that, clinically, there was no doubt about the case being one of psoriasis; he regarded it as sclerodermia with accidental psoriasis. Psoriasis, being so common, might co-exist with any skin lesion.

Dr. J. M. H. MACLEOD regarded the condition as psoriasis. He was familiar with psoriasis in association with joint trouble. The occurrence of sclerodermia in this patient might be a coincidence.

### Case of Lichen Planus and Syphilis.

By S. E. DORE, M.D.

THIS man, aged 65, was sent to me by Colonel Harrison from the venereal disease department of the hospital with a history of syphilis. He had two hard chancres on the frænum, and the *Spirochæta pallida* was found. He

received ten injections of "914," equivalent to 5.55 grm. A fortnight after the last injection a copious eruption appeared on the trunk, which somewhat closely resembled pityriasis rosea. Later, however, it became progressively less oedematous, and darker in colour, the individual lesions becoming flatter and shiny on the surface. At the same time that the eruption came out on the trunk, he was said to have had bullæ on the lips and inside the mouth. When I first saw him, on April 21, 1922, there were areas of denuded epithelium in these situations. There is now a severe and extensive eruption of lichen planus on the trunk and limbs, the papules in many parts having coalesced so as to form large plaques. The whole eruption is deeply pigmented, and there are well marked and extensive patches resembling leukoplakia on the tongue and buccal mucous membranes; he also has similar patches on the glans penis.

Dr. WHITFIELD, remarking on the eruption having a resemblance to pityriasis rosea when it first came out, asked whether any other members had been struck by the same thing. He recalled three cases in which he was trapped in the same way, and a fortnight later it became clear the disease was lichen planus. Some acute cases of lichen planus began with almost urticarial erythematous papules, with scales in the centre, and a little ring of erythema outside.

### Case of Parakeratosis Variegata in a Man, aged 60.

By S. E. DORE, M.D.

THIS patient was shown at the British Medical Association meeting at Newcastle last summer by Dr. Wells Patterson. He then had an eruption on the trunk and limbs, quite different from the present appearance; it was much pinker in colour, and blue in parts, and was striated and retiform in character. The diagnosis made at that time was parapsoriasis, but Dr. Heath and some other members regarded it as an instance of angioma serpiginosum. The patient has since been in the Edinburgh Royal Infirmary, under Dr. Cranston Low, from January to March, 1922. Dr. Cranston Low kindly wrote to me about him, and said he regarded the case as one of parakeratosis variegata of the type described by Radcliffe Crocker as xantho-erythrodermia perstans, and pointed out that the yellow colour became apparent on pressing the blood out of the skin. He had tried sulphur, salicylic acid, tar and chrysarobin without effect. X-rays and the mercury vapour lamp also failed to influence the eruption. Eight injections of sterilized milk were then administered intramuscularly into the buttocks twice a week, beginning with 2.5 c.c. and increasing to 10 c.c. No rise of temperature followed, but there was a marked leucocytosis for twenty-four hours after the injection, and there seemed to be a slight improvement in the eruption. A piece of skin was excised, but showed nothing unusual. When the patient was seen at St. Thomas's Hospital in June, 1922, he stated that he had become worse during the past eight or ten months, and complained of severe itching. The eruption had lost its original distinctive characters, and had become merged in a general redness affecting the scalp and face and the upper part of the trunk, leaving only the tips of the elbows, the palms and the legs free. The skin was of a deep red, almost crimson tint, was distinctly thickened and flaccid, and showed rugosities due to keratosis and scaling, the last feature being especially developed on the front of the chest and upper abdomen, the general appearance being comparable to that of the hide of an elephant. There

## 20 Dore: *Parakeratosis Variegata*; *Atrophic Lichen Planus*

was also considerable tenderness of the skin on pressure, but this became less noticeable at a later date. On physical examination at the hospital nothing was found except a definite enlargement of the spleen, which could be palpated beneath the costal margin. The differential blood count since submitted was as follows:—

Red cells ...	...	...	...	...	6,352,000 per cubic millimetre
White cells ...	...	...	...	...	8,400 " "
Hæmoglobin ...	...	...	...	...	90 per cent. "
Colour index ...	...	...	...	...	0.9 "

### *Differential Count of White Cells.*

Polymorphonuclear neutrophils ...	...	...	...	46 per cent.
Polymorphonuclear eosinophils ...	...	...	...	—
Polymorphonuclear basophils ...	...	...	...	—
Lymphocytes, small ...	...	...	...	36 per cent.
Lymphocytes, large ...	...	...	...	14 "
Large mononuclears ...	...	...	...	3 "
Myelocyte neutrophils ...	...	...	...	1 "

### DISCUSSION.

Dr. J. J. PRINGLE asked whether any member could say what happened to cases of parakeratosis variegata in the later stages of the disease. He had an impression that they did very badly, probably finding their way, eventually, into the workhouses to die there. He had not been able to follow up any such cases, and did not know of anyone who had done so.

Dr. GRAHAM LITTLE said the second of these cases recalled one over which he had been puzzled for three or four years. He had shown the case before the Section twice without receiving much help. There was the same general infiltration and the curious pigmentation. When he showed his own case the suggestion was that it was probably one of abnormal mycosis fungoides; but the patient had got a little better rather than worse. He at first thought it might be like Dr. Sequeira's erythrodermia; but in his own case there had been no change in the blood picture.

Dr. J. H. SEQUEIRA said he did not think this form of erythrodermia could be distinguished from the type Dr. Panton and he had described without a differential blood count. The essential feature in that condition was the persistent high percentage of small lymphocytes.

## Case of Atrophic Lichen Planus in a Woman, aged 40.

By S. E. DORE, M.D.

The interest of this case, I think, lies in its similarity to white spot disease or morphea guttata, several cases of which have been shown here. In that disease, however, the lesions chiefly affect the supraclavicular regions and back of the neck and shoulders. In this case they are situated on the front of the chest and in the suprapubic region, where there are small, flat, hard papules, some of which have a slightly erythematous border. There are also a few typical lichen planus papules scattered over the back and shoulders.

This appears to me to be an example of one of those cases of lichen planus atrophicus or sclerosus which have been confused with morphea guttata.

### Case showing Results of Treatment by Trepol.

By J. H. SEQUEIRA, M.D.

I HAVE brought this patient, a man aged 64, suffering from secondary syphilis, to show the pigmentation in the mouth caused by bismuth. He has been treated with "trepol," Levaditi's bismuth compound introduced for the treatment of syphilis. Its intensity depends on the degree of sepsis in the mouth. There is extensive blue staining of the buccal and gingival mucosa. The coloration is due to a deposition of sulphide of bismuth. The discoloration appears about a week after the first injection, and very soon after the first injection the patient complains of pain in the mouth, and there is some stomatitis with an offensive odour. Trepol seems to have an earlier effect on the blood reaction than on the clinical signs of syphilis.

I am indebted to Professor Bulloch, F.R.S., for the opportunity of using "trepol."

### Case of Lymphoblastic Erythrodermia.

By WILLIAM DYSON, M.D.

PATIENT, H. M., a male, aged 23, admitted as an in-patient to the Manchester Hospital for Diseases of the Skin, on June 13, 1922.

History: In June, 1919, whilst in France, an erythematous eruption appeared on the front of the chest. The eruption gradually spread, involving the whole of the trunk, face, and legs. It reached a maximum eighteen months ago, and since that date has remained stationary. When on leave he was isolated in the Grove Military Hospital for (?) German measles, and at the depot at Shrewsbury was under observation for suspected scarlet fever.

His general health is good, but he complains of intense pruritus, which is worse when he becomes warm and causes him to have sleepless nights. Appetite good; bowels constipated. In October, 1918, he suffered from trench fever. He was invalided out of the Army for neurasthenia.

Family history: Good, with no history of a similar condition in any of his relatives.

Condition on admission: Fair, red-haired, freckled, of good physique; has a general erythema, most intense over a band-like area surrounding the body, extending from just below the nipple line to the lower costal margin. Over the area the skin has the appearance of being slightly swollen and œdematous. In the groin and on the inner side of the upper arm in the region of the axillæ the rash is mottled in appearance, and purpuric. There is no desquamation, nor has there been any whilst he has been under my observation. There was marked dermatographia before admission, but this has now disappeared.

The lymphatic glands, both in the groins and axillæ, are distinctly enlarged, equalling the size of a hazel nut. Liver and spleen normal. Heart, lungs and urine normal. Teeth good. Tonsils not enlarged nor showing evidence of sepsis. Knee-jerks and abdominal reflexes exaggerated.

The patient is of a nervous temperament.

Blood-count (*July 5, 1922*): Red blood cells, 4,800,000; white blood cells, 8,800. Differential blood count: Polymorphs, 43 per cent.; small lymphocytes, 46 per cent.; large lymphocytes, 11 per cent. *July 13, 1922*: Red blood cells,

4,800,000; white blood cells, 13,000. Differential blood count: Polymorphs, 32 per cent.; small lymphocytes, 60 per cent.; large lymphocytes, 8 per cent. Sections of the skin show only the ordinary signs of inflammation.

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At a Special Meeting of the Section held July 20, 1922, Dr. T. C. GILCHRIST (Johns Hopkins University, Baltimore, U.S.A.) read a paper on "Some Problems in Dermatology" (illustrated by lantern-slides).



## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Two Cases of Multiple Carcinoma.

By J. H. SEQUEIRA, M.D.

*Case I.*—This is a woman, aged 57, single, who has been under my care, at intervals, since 1912, when she was sent to me by Dr. Jekyll with a rodent ulcer of the left frontal region of the ordinary type, rather superficial, and tending to cicatrize. Although I have seen her at intervals and her doctor has seen her very often, she never mentioned that she had any other lesion, except patches of psoriasis on her elbows and knees; but for twenty-five years she has had, on the left upper abdomen, a patch which she herself thought to be psoriasis, and which at one time was covered with a considerable scale. Under treatment by X-rays and ointment, much of this scaling has been removed, and now she has an irregular area, 3 in. by 2 in. infiltration, with a red, rather vascular surface, though there has been no hæmorrhage, and practically no discharge from the surface for a long time. It is a condition which I originally described in my notes as "Pagetoid," i.e., resembling Paget's disease of the nipple. I have no sections of it.

*Case II.*—The patient is an old man aged 74. His present lesions have, as far as he knows, been in existence for thirteen years. In August, 1921, he had radium treatment applied to a rodent ulcer above his right eye, which is now soundly scarred. He presents eight lesions; the largest is mid-sacral, measures 2 in. by 1½ in., irregularly shaped, of purplish-red colour, having fine silvery scales on its surface and showing small brownish crusts of dried blood, varying in size from that of a pin-head to that of a millet seed. Above and to the left of this is a small similar lesion, the size of a pea; 1½ in. higher and to the left of the mid-line is a well defined purplish smooth lesion about the size of a shilling, its edge not appearing to be raised. This and all the lesions are freely movable, not tender, thin and easily pinched up. There is a fourth patch in the mid-dorsal region to the left of the spine similar to that last described, and a fifth smaller lesion the size of a threepenny-piece to the right of the second dorsal spine. In front of the chest are three patches, one in the middle of the sternum, the size of a florin, with a pink scaly surface, a similar slightly scaling area over the xiphisternum, and two small pale patches, one over the second left costal cartilage and two below the middle line of the left clavicle. This last patch is yellowish-brown in colour, oval in shape, and consists of confluent, finely scaling, palpable, small papules. The whole area is slightly raised 1·2 cm. in diameter and has a faintly pink tinge in its middle. This was entirely excised for microscopical sections and showed marginal downgrowths

of basal cells with a perfectly regular border. A second section taken from a brownish oval nodule on the front of the chest showed a definite, typical appearance of basal-celled carcinoma.

I am showing these cases of multiple carcinomatous lesions of the skin in order to initiate a discussion as to whether Bowen's carcinoma is a clinical entity or whether it is to be recognized as one of the many appearances of pre-cancerous and cancerous conditions found in cases of multiple carcinoma cutis.

### Case of Multiple Superficial Rodent Ulcer ; possible Embryonic Sweat-duct Origin.

By H. G. ADAMSON, M.D. (President).

THIS patient presents a type of superficial multiple rodent ulcer to which the attention of this Section has lately been called by the exhibition of several similar cases. Three such cases were shown at three consecutive meetings in 1920 by Dr. Graham Little, Dr. Agnes Savill, and Dr. Gray,<sup>1</sup> and Dr. Little more recently brought up three cases to the meeting in March when Dr. Darier

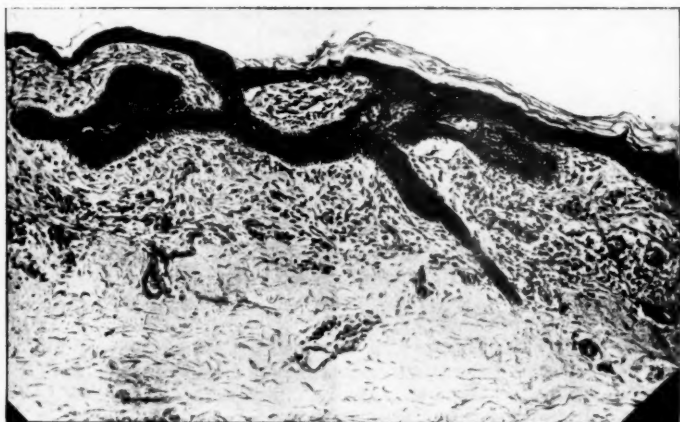


FIG. 1.

was present.<sup>2</sup> Dr. Darier identified them with a baso-cellular superficial epithelioma which he had described as "Pagetoid" and distinguished them from Bowen's carcinoma.

The patient, E. C., has five lesions, one on the back and three on the abdomen and one on the front of the left thigh. They show the usual irregularly circular discoid patches, with scarcely any depth or infiltration, covered with fine scale-crusts, and with a very narrow waxy rim or "rolled edge," and a tendency to resolve by fine atrophic scarring.

<sup>1</sup> *Proceedings*, 1919-20, xiii (Sect. Derm.), pp. 30, 54, 68.

<sup>2</sup> *Ibid.*, 1922, xv (Sect. Derm.), p. 36.

The particular interest of this case, and my reason for exhibiting it, consist in the microscopical appearances. The microscopical sections show the flattened button-like masses of basal cells springing from the basal-cell layer of the epidermis. A feature to which I particularly wish to call attention is the fact that several of these bud-like processes are continued into long narrow tubules of basal-cell structure which are indistinguishable from sweat-ducts. This feature seems to me to confirm an opinion which I have previously expressed, that while rodent ulcers are usually embryonic pilosebaceous

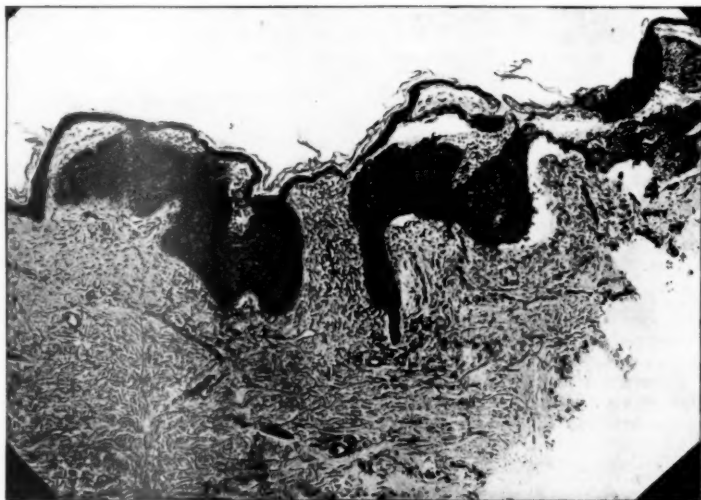


FIG. 2.

follicles or basal-cell growths from the epidermis which were destined to become pilosebaceous follicles but have lost their power of differentiation, they may be sometimes of embryonic sweat-duct origin, that is to say, they are buddings from the basal-cell layer of the epidermis which, if they had occurred in the embryo would have become sweat apparatus, but which occurring at a later period have lost their power of differentiation and retained only that of proliferation. In other words they are buddings from the basal layer which are trying to become sweat-apparatus.

#### DISCUSSION.

Dr. GRAHAM LITTLE said he thought these three cases must be placed in one group; equally certainly, he thought, they were of the same type as the series of cases to which the President had alluded. He thought he (Dr. Little) was the first to show this type, in a case he exhibited about two years ago, the patient having a very large number of lesions, almost all of the flat type, but one, on the neck, of the large warty rodent ulcer type. When he showed the case, most of the Members present were inclined to question the diagnosis until they saw sections, and the view that it was lupus erythematosus was largely expressed. That was of great interest because the cases of this type were almost uniformly of an erythematoid surface-colour, and the

erythematoid aspect of all these cases was, clinically, a very remarkable differentiating feature of the group. He thought the type "erythematoid" should be added to the group, to distinguish it in that way from other varieties of growth. He had been able to see Dr. Fordyce a few weeks ago, and had a talk with him on this subject. Dr. Fordyce had interested him very much by saying that he had had a series of these cases which he (Dr. Fordyce) had also diagnosed as lupus erythematosus, and that the rodent histology had astonished him very much. He (Dr. Little) agreed with the President that there was always clinical evidence of a rodent ulcer in the edge of the rodent patch; in these cases now present a very minute but definite edge was discernible. It was a special character of this group that the lesions showed little or no malignancy, and that was borne out in his first case, for several of the lesions had undergone spontaneous atrophy. This was also supported by Dr. Pringle's statement at the meeting at which Dr. Darier was present, in which Dr. Pringle described a case he had seen of a gentleman, aged 83, who had had one or more lesions of that type since boyhood. With regard to identification with Bowen's disease, Dr. Darier saw the three cases of this variety which were brought up when he was here, and, when asked the question, he very definitely said they were not Bowen's disease. Another very interesting feature about one of his (Dr. Little's) personal cases was that psoriasis was noticed during the eruption of rodents. That was present also in both Dr. Gray's cases. In Dr. Agnes Savill's case the eruption of rodents had apparently followed on the site of extensive seborrhœa of the skin. It was remarkable that in so many of these rare cases there had been some preliminary disease, such as psoriasis or seborrhœa, before the rodent ulcers appeared.

Dr. ARTHUR WHITFIELD said that many years ago he had shown, before the Dermatological Society of London, a case of rodent ulcer of the shoulder, which he had observed developing in a patch of psoriasis. That patient had generalized psoriasis, and he saw it before the rodent ulcer came. But that was not of this type now shown; it was typical rodent ulcer. At that meeting he raised the question whether, in view of the well-known fact that prolonged arsenical medication would set up epitheliomatous changes in the skin, it was possibly one of the reasons why it occurred in this patient, who, to his knowledge, had for a long time been taking large doses of arsenic, at several periods of her life.

Dr. W. J. O'DONOVAN said this could not be a new disease, and it would be a pity if dermatologists were pinned down to a label of multiple rodent ulcer, because in Dr. Sequeira's case there were conditions of pre-carcinoma, multiple rodent ulcer, and squamous carcinoma present together. He did not think they could recognize these as rodent ulcers by their appearance.

Dr. A. M. H. GRAY did not agree with what Dr. O'Donovan had just said; he regarded the condition under discussion as a definite one, both clinically and microscopically. That one of the lesions of Dr. Sequeira's published case showed changes of squamous epithelioma type did not alter the main features of the cases, because sometimes lesions of the rodent ulcer underwent changes indistinguishable from those of squamous-celled epithelioma. Most of these cases had been microscopied, and had shown a uniformity in microscopical appearance. With regard to Bowen's disease, he agreed with Dr. Graham Little. He did not think there had yet been seen in this country a case which was identical with the type described by Bowen, whereas Darier certainly had seen such cases and had also examined them microscopically. This gave great value to the opinion of Darier on this matter. He thought it was high time that this disease was properly described and published, as there existed considerable confusion of thought about it.

Dr. SAVATARD said he had hoped to show a patient, whom, on Darier's authority, he regarded as a case of Bowen's disease. The type was so different from those shown that afternoon that he thought dermatologists were justified in dogmatically differentiating one from the other. Several members had already emphasized the fact that the cases seen that day had a decided edge; he did not think a decided edge had been demonstrated in Bowen's disease, nor in Paget's disease. The real differential diagnosis

was as between Bowen's disease and Paget's disease. He would have diagnosed this case as extra-mammary Paget's disease rather than as a Bowen's if he had not looked up to see what Darier said on the subject. That authority pointed out how similar histologically the two conditions were, but he stated, with great clearness, that there was an important histological difference—namely, that the hyperkeratosis so characteristic of Bowen's disease was made possible by the retention of the filaments of union between the rete cells, whereas in Paget's disease the dissolution of these filaments prevented the formation of coherent horny layers. In his case, of which he showed photographs, he first diagnosed verrucose lupus, because of the keratosis. He therefore scraped it. It remained sound for two or three years, after which there was a recurrence, and the patient returned last month. He then excised it, and found what he regarded as Bowen's disease. Bowen and others had emphasized the difficulty of differentiating this disease from tertiary syphilis. The photograph he handed round suggested tertiary syphilis, but the man had no syphilitic history, and his Wassermann reaction was negative. The lesion had already become malignant. He agreed with Dr. Gray that these cases of superficial rodent ulcer should be grouped together and some distinctive name applied to them. He did not like the name "superficial rodent."

Dr. MACLEOD said that he thought the superficial type of rodent ulcer, several cases of which had been shown, was different from Bowen's disease. He did not think he had ever seen a case of Bowen's disease except one which had been diagnosed as Paget's disease. Bowen made a definite point of the presence of dyskeratosis, and he (Dr. MacLeod) submitted that some of the cases which had been described as Paget's disease, elsewhere than on the nipples, might probably have been Bowen's pre-cancerous condition.

### Case of Lupus Erythematosus associated with Lichen Planus.

By GEORGE PERNET, M.D.

THE patient is a woman, aged 64. She has two patches of lupus erythematosus on the left side of the face, and a small recent patch on the bridge of the nose. They began to appear seven years ago. But it must be noted that the rims of both ears are atrophied and irregular. On the left side of the neck there are some old scars of tuberculous glands dating from childhood. In addition, there is a palm-sized area of lichen planus verrucosus on the right leg, also dating from seven years back. The points of interest in this case are: (1) The association of the two conditions of about the same duration; (2) their unilaterality; especially in the case of the lupus erythematosus.

### Case of (?) Pellagra.

By HUGH S. STANNUS, M.D.

THE case now shown is one which, if it had occurred in an endemic area, would have been diagnosed without doubt as one of early pellagra. I am anxious to elicit the opinion of the members of this Section as to whether the lesions presented could be explained on any other pathogeny. The man is chief storekeeper to the Nigerian Railways, and went to West Africa some fifteen months ago for the first time. Three or four months after his arrival he developed an acute dermatitis, involving the exposed portions of his hands and wrists and neck and lower part of the face. Bullæ formed, and, later,

## 28 Stannus: (?) Pellagra; Whitfield: Demodex Impetigo

scaling took place. The areas involved are strictly those exposed to sunlight according to his dress and head covering. He shows other slight but suggestive symptoms: there is some disturbance of the epithelial covering of the tongue with a resulting leucoplakia-like condition in some areas, and in others some slight denudation; there is also some heaping up of white sodden epithelium at the muco-cutaneous junctions at the angles of the lips, and some discoloration at the external canthi, signs to which a good deal of importance is attached. He has suffered from dyspepsia and constipation, and became rather depressed about the persisting condition of his hands, but has no frank nervous symptoms. Before going to Africa he had never been out of Europe. He states that he had always had a skin sensitive to sunburn.

### DISCUSSION.

Dr. PERNET said he had seen pellagra in Italy, and one or two cases in London, and this case did not somehow seem to fit in with that diagnosis, but might be due to sun exposure in West Africa.

Dr. WHITFIELD said that although he had seen a number of cases of solar disturbance of the skin in Colonials, the condition was not like that of this patient; there was none of the smudgy pigmentation with hyperkeratosis at the back of the hands which was seen in solar cases. What struck him as characteristic of pellagra was the extremely defined edge, which was never seen in Colonial cases of sun disturbance. The patient had a very smooth skin, with a definite edge, and a little line of pigment beyond.

Dr. H. G. ADAMSON (President) said that the so-called solar skin only developed after years of exposure: it was not an immediate effect of strong sunlight. On the fingers the condition looked like lupus erythematosus.

Dr. STANNUS (in reply) said that the descriptions of pellagra in the text-books were often very misleading: they appeared to have been copied from one book to another, and were mostly descriptions of full-blown cases of many years' standing. The features of the present case were all characteristic, and he agreed with Dr. Whitfield that the condition known as "sailor's skin" was only produced after many years; if there were no other hypothesis offered in explanation, he believed it to be a case of pellagra. As regards the sensitiveness to sunburn, he believed it was no more than is commonly seen among individuals of the same degree of fairness.

## Photographs of Demodex Impetigo.

Shown by A. WHITFIELD, M.D.

A FEW meetings ago I showed photographs of a case of impetigo of bullous nature, in which I discovered the demodex, and I found the condition had been described in Australia. Since then I have had a considerable number of cases. All my earlier cases were at the age of puberty, or later, i.e., at an age when demodex may be found in the skin. Therefore I have been on the look-out for a much earlier stage. Recently a child, aged 15 months, was brought to my out-patient department with an extraordinary bullous impetigo, and I said it would be worth while to look for the demodex in it, and it was found. Demodex has not been described on the skin at 15 months of age, and at that age, unless camphorated oil has been rubbed in, the children have no comedones. This case, therefore, is evidence that there is a definite association between the demodex and impetigo in some instances. The fact that I am



now able to prophesy the cases in which one is likely to find the demodex appears to me to be evidence that it has some causal relationship in the production of the disease or in the development of special features.

### **Case of Sclerodermia.**

By HALDIN DAVIS, M.B., F.R.C.S.

PATIENT, a woman, aged 56, presents a condition of the skin which I have called sclerodermia, although it is not quite like an ordinary case of that disease. The whole skin of the trunk, extending from the clavicles to the tops of the thighs, is unnaturally smooth and of the consistency of hard wax. It reminds one of a case of sclerema neonatorum. The patient finds that it makes it very difficult for her to stoop owing to its stiffness. She also complains bitterly of the severe pruritus which it causes. The condition dates from about nine months ago, when she was wearing a ring pessary, which she neglected to change for several months. It appears probable that the cutaneous disorder has been caused by septic absorption due to the presence of this foreign body for too prolonged a period in the vagina. Other examples of sclerodermia due to septic absorption have been published, but as a rule due to septic teeth. These cannot have been the cause in this patient, for she had all her teeth extracted some years ago.

#### **DISCUSSION.**

Dr. GRAHAM LITTLE regarded the case as diffuse sclerodermia. Septic absorption of diffuse sclerodermia was present in a case of his own, which he had shown before the Section. That patient had a large part of the body immobilized by sclerodermia, so that she could not feed herself, and she had to be carried up and down stairs. The condition was advancing. The whole of the affected areas cleared up astonishingly with the removal of all her teeth, many of which were septic. She was now earning her living by type-writing, the last thing of which one would have thought her physically capable.

Dr. G. PERNET said he considered this was a typical and characteristic case of sclerodermia. Many years ago he saw a similar condition in a middle-aged clergyman, whose abdomen and other parts of the trunk were completely sclerosed. In the patient now shown the appearances about the nipples were of interest, as those areas had escaped involvement.

### **Case of Angiokeratoma.**

By HALDIN DAVIS, M.B., F.R.C.S.

PATIENT, a girl, aged 17, presents a very striking example of angiokeratoma on the hands. They first began to be affected about seven years ago and have become steadily worse year by year. There are numerous typical lesions on all the fingers of both hands, small bluish swellings deeply imbedded in the skin and surmounted by thin horny caps. All the digits are fat and puffy, the circulation is obviously very sluggish and during the winter she suffers greatly from chilblains. The condition is a very serious handicap to her as it prevents her from doing any ordinary work.



## DISCUSSION.

Dr. F. PARKES WEBER said that some years ago<sup>1</sup> he showed a case of severe angiokeratoma of the hands, in which the fingers and toes, on Röntgen-ray examination, showed bone-changes somewhat resembling those occurring in sclerodactylia. These bone-changes had since increased. He thought that the so-called angiokeratoma of the scrotum was to be separated decidedly from angiokeratoma of the hands.

Dr. H. G. ADAMSON (President) said that the classical case of this disease was the one Dr. Pringle described, with a coloured drawing,<sup>2</sup> almost exactly like this patient's hands. It was a characteristic affection, and occurred in people with a chilblain circulation. It was very common in Italy. There were dilated vessels, and they formed little cysts in the skin. The hyperkeratosis was a secondary condition over the top of the dilated vessels.

Dr. A. EDDOWES said that two essential points should be kept in mind in the treatment. The first was to try to soften the warty collections of epithelium, and the second was to help the circulation artificially. It would be useful for this girl to wash her hands at night in warm water, and afterwards rub into them cold cream containing boric acid to remove the horny accumulations, and to wear gloves in cold weather. The hands should also be massaged.

**Case of Xanthoma Diabeticorum.**

By J. H. SEQUEIRA, M.D.

(Shown by W. J. O'DONOVAN, M.D.)

PATIENT, a male, aged 25, was discharged from the Army in 1919 with diabetes and has since been under Dr. Leyton who has been treating him dietetically since 1921. He shows well-defined small oval or rounded flat xanthoma lesions arranged in lines along the flexion creases of the hands. There are similar nodules arranged in groups over the knuckles and elbows and on the dorsum of the feet. The lesions have lessened since he has been dieting himself strictly. At the meeting of the Dermatological Association in London last year I showed a woman who had ten times the normal amount of cholesterin in the blood and who showed similar xanthoma lesions, but this man's blood, tested in the same laboratory, showed slightly less than the normal percentage of cholesterin in the blood.

**Epithelioma Adenoides Cysticum.**

By LOUIS SAVATARD.

UNDER the title of "Epithelioma Adenoides Cysticum," Brooke [3], in 1891, described benign growths of the skin which till then had not been properly appreciated by dermatologists. His paper was communicated to the Manchester Pathological Society and was published in the only published *Transactions* of that Society (1891-1892). Later it was published in the *British Journal of Dermatology* (September, 1892).

At about the same time Fordyce [9], of New York, was carrying out similar research and communicated his results to the sixteenth annual meeting

<sup>1</sup> F. Parkes Weber, "Angiokeratoma, with Bony Changes," *Proc. Roy. Soc. Med.*, 1914, vii (Clin. Sect.), p. 25.

<sup>2</sup> *Brit. Journ. Derm.*, 1891, iii, pp. 237, 282, 309.

of the American Dermatological Association, New London, in September, 1892, under the title of "Multiple Benign Cystic Epithelioma of the Skin," and his paper was published in the *Journal of Cutaneous and Genito-Urinary Diseases* (December, 1892). Fordyce's clinical and histological descriptions were practically identical with Brooke's.

Before this, however, Perry [17] had published a beautiful example of this condition under the title of "Adenoma of the Sweat Glands" in the "International Atlas of rare Skin Diseases" (No. ix), but Balzer and Ménétrier's [2] case of "Sebaceous Adenoma of the Face and Scalp," reported in the *Archives de Physiologie* (1888) is probably the first recorded case of Brooke's disease. Their clinical and histological pictures are identical with those of Brooke and Fordyce. Many cases have been recorded in recent years and the literature on the subject is now by no means inconsiderable. Before, however, reviewing this we will refer to Brooke's original communication, the result of investigation in four cases, a mother and her two daughters and a young woman aged 20. Brooke says:—

"The affliction presents itself in the form of small tumours varying in size from a pin's head, projecting very slightly above the surface, to that of the half of a small pea. They are at first of the colour of the surrounding skin or may be a little darker. In a few isolated lesions on the trunk black dots could be seen beneath the epidermis. As the growths increase in size they become often shining and translucent, but hardly sufficiently so to suggest that they contain fluid. Some have a faint yellow or bluish tinge, nearly all contain one or more white milium-like bodies. The size of these bodies bears no marked relation to that of the lesion in which they occur. The tumours are firm without being hard and, if taken between the fingers, can be felt to lie in the skin and to move with it. As regards their distribution the sites of predilection were the space between the eyebrows, the root of the nose, the nostrils and neighbouring area of the cheeks, the upper lip and, to a less extent, the chin. In these situations they become so thickly grouped together as to form raised lumpy patches of most disfiguring appearance. They also occurred on the back, on the scalp and on the ears. They were strewn thickly over the shoulders, on the neck and upper part of the arms and slightly on the chest. The lesions on the scalp were as copiously covered with hair as was the normal skin around them.

"The course of the affection is always slow, but may vary at times, taking on a sudden acceleration, even after many years' duration.

"It begins in youth, for the most part between the tenth and fourteenth years. In no case which has yet been recorded has there been any attempt at spontaneous involution noted; on the contrary, the lesions either persist unchanged for years, or increase until they reach the size of a small pea, a limit which they never exceed."

Fordyce's description was practically identical with that given by Brooke. He, too, notes that black pigment spots were found intermingled with the lesions. In some he noted an apparent central depression which led them to simulate very closely the lesions in molluscum contagiosum. Perry's case also showed black dots in a few of the lesions, and histologically many of the cells of the "gland" were loaded with brown pigment. Balzer and Ménétrier make no mention of pigment.

(I) Brooke's Cases [3].

Mrs. E., aged 50. The lesions were first observed in her youth and she had only a very few when she was married at the age of 21. Of late years they have much increased in number.

L. E., aged 18, presented numerous new growths, which agglomerate in masses in the region of the face most affected in the mother, but are also scattered freely over

the neck, upper part of the back, shoulders and arms, and very sparsely on the chest (fig. 1, p. 34).

L. E., aged 14, presented comparatively few nodules, but the majority of these are quite well developed. They are limited to the face and neck. As was the case with her sister, they were first discovered when she was about 10 years of age, and have been gradually increasing in number and size.

M. C., a girl, aged 20. The tumours were first noticed eight years ago between the eyebrows, and had increased very slowly until about two years ago when a more rapid increase had taken place. They are distributed about the eyebrows, inner corner of the eyes, bridge of nose, neighbourhood of the nostrils and mouth, a few discrete lesions being scattered about the rest of the face. There are some on the neck, at the nape and sides, and a larger number on the upper third of the back and shoulders, but more especially in the space between the scapulae. Family history negative.

(II) *Fordyce's Cases* [9].

Daughter, aged 19. The eruption was first noticed on the left temple and forehead six years previously. During the past two years there appears to have been no increase either in the number or size of the lesions. The tumours are scattered over the forehead, temples, eyelids, cheeks, nose, behind and below the ears, back of the neck, and through the hair. In the interclavicular regions from fifteen to twenty tumours are seen and there are a few over the upper portion of the chest.

Mother. In size, appearance, and general distribution the lesions were almost the counterparts of those on the daughter's face. First noticed at the age of 15. (Her father had a group of tumours like these on her daughter's temple and in the same locality.) The eruption extends over the forehead, face, ears, neck and nose and over the upper portions of the back and chest and has existed for over thirty years.

(III) *Perry's Case* [17].

A woman, aged 31, single. The lesions were noticed on the right side of the nose at 10 years of age. At the age of 22 "pimples" were to be seen in their present situation, but had increased in number and size during the last year. There were a few on the scalp but the rest of the body was unaffected. The family history was negative.

(IV) *Balzer's and Ménétrier's Case* [2].

A woman, aged 21. Lesions on the face, scalp and neck. Onset at 11 years of age. Her father is said to have been similarly affected.

(V) Jarisch, in 1894 [12] reported a case in a young man aged 22, who presented several lesions on the face, some of which were scabbed, giving the appearance of an impetigo. The onset dated from puberty. The histological illustrations suggest that this is a true case in spite of the ulceration of some of the tumours, but this ulceration, however, does not necessarily imply malignancy, as I hope to demonstrate later.

(VI) Pick, in 1901 [18] described a case in a man, aged 45, who, since he was 8 years of age, had suffered from severe "acne rosacea" of the forehead, nose and cheeks, which were red and presented numerous small lesions of rosacea. Associated with those on the left malar region there was a peculiar patch about the size of a shilling, over which the blood-vessels were dilated and which had a covering of small adherent scales resembling those of lupus erythematosus. Several small lesions of a similar character were present on the right malar region, forehead and above the right eyebrow.

(VII) Dubreuilh and Auché, in 1902 [8] reported a case of multiple benign epitheliomata of the scalp in a woman, and Adamson later demonstrated its identity with the condition under review.

(VIII) Csillag [5], in 1906, reported cases in a mother and daughter.

(IX) Pusey in his text-book (1907) [19] illustrates two typical cases of the multiform variety in father and daughter. The lesions are apparently limited to the face, ears and neck. No history of these particular patients is given, though the author remarks with regard to the absence of malignant supervention, "My elder patient, a man well beyond 70, in whom, regardless of his condition, an epithelioma would not have been a surprise."

(X) Heidingsfeld, in 1908 [11], recorded cases in a man, aged 65, and in all his children—two sons, aged 34 and 30, and two daughters, aged 38 and 36, and a history of the same affection in his maternal uncle and aunt, though neither his mother nor father was similarly affected. The growths in these cases were first evident between the twenty-fifth and thirtieth years.

So far in the cases under review the lesions have been multiple, but Wolters [28], in 1901, recorded a solitary lesion in the case of a woman aged 20, who presented on the right eyebrow a "yellowish red-coloured lint-seed-sized tumour," which had been present since birth and which, histologically, was identical with Brooke's disease.

(XI) Sutton, in 1911 [21], cited a remarkable case in a negro woman, aged 60. The growths which varied in size from "the head of a small pin to the tip of a navy bean," were darker than the normal skin and were distributed over the face, forehead, malar regions and sides of the neck. White, of Boston, who examined the specimens suggested that the growth was more nearly a so-called tricho-epithelioma than a typical representation of the Brooke-Fordyce type of disease. These tumours were greatly treasured by the family in spite of the fact that the patient's mother, three of her mother's sisters, her own sisters (nine), her own brothers (two), all her own children (fourteen), her surviving sister's children, and her brothers' children, all showed similar lesions on the face.

One of my own cases somewhat resembles this case, but has no such remarkable family history.

(XII) Adamson [1] showed before this Section, in February, 1914, a most interesting series of cases in a mother, two sons and a daughter. The communication was reported in the *Proceedings of the Royal Society of Medicine* for 1914, and is accompanied by several clinical and histological illustrations, one of which (fig. 10) is another instance of a solitary lesion. It was situated in the lower lid of a man, aged 37, "who now had a typical rodent ulcer of two years' duration in the nasolabial furrow."

(XIII) Miller, in 1915 [14] recorded three cases in a brother and his two sisters. The father and another brother were said to be similarly affected.

(XIV) Adamson, in 1918 [1] reported a case of multiple benign basal-cell epithelioma of the scalp. The patient, a man aged 61, presented on the scalp from forty to fifty tumours which varied in size from that of a hemp-seed to that of a chestnut. They were of the colour of the skin with smooth surface devoid of hair, firm and movable on the skull. They had been noticed for twelve years, but there had been similar tumours on the back for forty years. Adamson established its identity with Dubreuilh and Auché's case and with the case of one of the sons affected with epithelioma adenoides cysticum, which he had reported previously and to which I have already referred.

(XV) Norman Paul [16] in his book, shows a typical example in the case of a woman, and we are informed that her grandfather, father and two brothers and a sister were similarly affected.

(XVI) Withers and Coleman, in 1920 [24] reported a case of multiple benign cystic epithelioma associated with xeroderma pigmentosum, but I cannot accept it as a true case of epithelioma adenoides cysticum (Brooke).

I shall now show you cases which have come under my own observation, and the first to which I shall direct your attention is one of Brooke's [3] original cases whom by chance I saw in my out-patient department last summer.

(i) She (fig. 1) was the elder daughter of the mother and two daughters referred to previously, now aged 49; and here I must report that Brooke's family record was not strictly accurate—instead of "a widow and her two only children" you should read "a mother and two of her daughters." For, though the mother died two years later her husband still survives, and they had in all nine children, six daughters and three sons. None of the sons was affected but the third, fourth, and later the fifth daughter developed well-marked evidence of the disease. The fourth daughter I have not been able to trace. She is married but has no children and has not communicated with other members of the family for years. I understand that the growths in her case have not attained the extravagance of her elder sister's. None of the grandchildren shows any evidence of this affection. You will see from the photograph (fig. 2) before you the great ravages the condition has made during the last thirty years.



FIG. 1.—Case I, aged 18.

The tumours have progressed in numbers and size. Between the brows, and on either side of the *ala nasi*, they have coalesced "to form raised lumpy patches of most disfiguring appearance" while several of the lesions on the back have attained the size of large cherries. This patient presented lesions all over the face and ears, numerous small ones on the scalp, neck, shoulders and chest, several minute ones on the forearms, none on the upper arms, and one—the size of a pea—is evident below the left calf; while the central third of the back is studded with tumours of varying sizes (fig. 3, p. 36). Numerous milia can be seen in and between the tumours on the face and a few of the growths show pigmentation beneath the epidermis. None of the tumours have ulcerated and none have involuted. The affection was first noticed at the age of 10. In spite of her disfigurement I have been unable to persuade the patient to attend for treatment. The condition is not, however, so evident to the eye as to the camera, because the lesions for the most part are the colour of the skin.

(ii) The next case is that of this patient's younger sister, now aged 41, who presented no lesions till she was 20 years of age. She was the sixth child and the fifth daughter of Mrs. E. The lesions are confined to the face. They slowly progressed up to a few years ago and seem to be stationary now. The distribution is typical of a case of moderate intensity.

Case (iii) The next case is that of Miss A, aged 34, in November, 1915, when she first consulted me. She presented numerous tumours on the face, neck, shoulders, scalp and back. They were for the most part of the colour of the skin. Some few, on the face only, included one or more milium-like bodies and some half dozen on the face and back



FIG. 2.—Case I, aged 49.

contained sub-epidermal pigment. One on the back was the size of a cherry (fig. 8, p. 43). The scalp tumours were all small and bereft of hair. While some were superficial others were fairly deeply embedded in the skin, two-thirds of the growth being beneath the skin's surface. The growths for the most part were firm without being hard and could be felt to lie in the skin and to move with it. The affection was first noticed during childhood. For the last seven years they have increased very slightly in number and size. The patient has six brothers but no other member of the family is similarly affected.

The next series of cases show a marked hereditary factor.

The mother, three daughters and a son of a family of nine were affected, and the third child (a son) of the eldest affected daughter presents an early solitary lesion.

The mother is dead but her photograph shows the characteristic lesions in the central third of the face. They had appeared at an early age but had not progressed in number or size after her marriage. These cases I first saw this year. They are not of the extensive type and the lesions are limited to the face.

(iv) The eldest sister, aged 45, presents pronounced milia inclusions and a few pigmented lesions. The tumours were not evident before her twenty-fifth year and are progressing very slowly.

(v) The brother, aged 42, presents a similar appearance. Most of his tumours date from boyhood and they are apparently stationary.

(vi) The second sister, aged 29, presents smaller lesions with fewer milia and no black dots. In her case the affection has only existed for five years.



FIG. 3.—Case I. Lesions of central third of back.

(vii) The third sister was brought to me only within the last few weeks and her case is so slight and the lesions so small, though characteristic, that I could not have made the diagnosis without the family history. She is aged 25.

(viii) The eldest sister's third son, aged 12, presents one small tumour on the right upper lid and a few milia without tumour formation on both upper lids.

The three cases following are instances of solitary lesions:—

(ix) The first, a girl, aged 11, whom I saw in 1912, presented on the right side of the bridge of the nose a skin-coloured, raised, translucent-looking tumour of fairly firm consistency, movable with the skin and apparently not invading the derma to any



great extent: in short, a tumour which, clinically, was indistinguishable from a non-pigmented mole (fig. 4). There were no milium-like bodies evident and none was present elsewhere on the face. The growth was first apparent five years previously and had progressed slowly. No other member of the family was similarly affected and no fresh lesions had appeared up to two years ago. Microscopically the section was almost identical with Brooke's [9] original drawings (fig. 5, p. 38).

(x) The second, a woman, aged 54, in 1917, presented a similar, though larger and more indurated, tumour in the same situation. The growth had appeared in childhood. There were no milia present. The family history was negative and no other lesions have since developed.



FIG. 4.—Case IX. A solitary lesion.

(xi) The third, a woman, aged 42, in 1916, presented in the left nasolabial furrow a small tumour which had existed since childhood. It had recently become ulcerated [20]. On examination I found an infective ulceration of a benign epithelioma.

(xii) My next case, which bears some resemblance to Sutton's case, is that of a girl, aged 15, who was sent by Dr. Horsman McNabb this year and who presented various small tumours on the face and neck. For the most part they did not exceed the size of a pin's head, though one, which had become ulcerated and scabbed on the right lower lid, was the size of a pea. The tumours of the upper lids were hardly raised above the surface of the skin, whilst those on the lower lids at the ala nasi and on the neck were raised above the skin, had a translucent appearance, and were the colour of the skin. The ulcerated tumour and the one beneath the left lower lid were firm to

the touch whilst the others were quite soft. The right lower lid tumour was clinically indistinguishable from a rodent ulcer and the one at the right ala nasi had a small vessel coursing over its surface and it simulated a non-pigmented mole. I thought that probably we had here different types of tumours. On histological examination I found them all apparently of basal-cell origin, though, as we shall see later, the upper lid tumours suggest a possibility of their being tricho-epithelioma. No white nor black dots were evident in the growths though a milium was present on the left upper lid and there were some pigmented naevi on the face. The tumours were first noticed two years ago. No other member of the family was similarly affected.

Later examination of the rodent-like tumour of the right lower lid shows that it has undergone a malignant development, and a recurrent nodule, after excision of the original ulcer, shows this development more clearly. Here,



FIG. 5.—Case IX. Microscopic section of a solitary lesion.

then, we have evidence that these benign growths *may* exceptionally become malignant. Cranston Low writes me of a similar case of his in which one tumour alone underwent malignant transformation.

My last two cases are further instances of solitary tumours. They both came to my out-patient department last month for some other affection.

(xiii) Mrs. R., aged 63, presented on the middle of the scalp a large lobulated tumour of the colour of the skin, its surface devoid of hair, smooth and shining. The growth, not apparently invading the deeper portion of the derma, was very constricted at its base and was nearly seven inches in circumference. It was firm without being indurated and was movable with the skin. There were no subjective symptoms. It had first appeared twenty years ago.

(xiv) Miss B., aged 26, presented a small mole-like tumour on the upper lip. Though I could not clinically make a positive diagnosis, the appearance, on close inspection, of loculi (not milia) beneath its surface suggested that the growth was not an ordinary epithelial mole. Histological examination proved that my surmise was correct. This small tumour was of the same duration as the preceding one. In both cases the family history was negative.

Summarizing the clinical aspect of this condition we find that it presents itself in the form of tumours, projecting very slightly above the surface, and varying in size from that of a pin's head to that of a pea, though on the scalp and back they may later attain the size of a walnut or even larger, and in these situations the major part of the growth may be beneath the skin's surface. They are at first the colour of the surrounding skin or may be a little darker. Many lesions on the face present one or more milium-like bodies (white dots) within them, whilst a few lesions show pigment (black dots) beneath the epidermis.

As these growths increase in size they often become shining and translucent. They may acquire a faint yellow or bluish tinge and many of the older tumours present a dusky reddish appearance due to minute vessels coursing over their surface. The tumours are firm without being hard and if taken between the fingers can be felt to lie in the skin and to move with it. In a case of moderate intensity the sites of predilection are between the brows, the root of the nose, the nostrils and neighbouring area of the cheeks, the upper lip and the chin, or, in other words, the central third of the face. In the more extensive cases they may cover the face; be profusely scattered over the scalp, neck, shoulders, chest and upper extremities; be thickly studded on the central third of the back to just below the waist. The older tumours on the scalp are bereft of hair.

Exceptionally the tumours may appear on the scalp alone. Or the tumours may be *solitary*, when they are clinically indistinguishable from the non-pigmented moles, and I would hazard the opinion that in course of time we shall find the solitary lesion the rule and the multiple form the exception.

The growths are often associated with other tumours of congenital origin.

Though women have provided the majority of cases the lesions are not infrequently met with in men. The age of onset is usually about puberty, occasionally in early adult life and rarely in middle age. The course of the affection is slow but may vary at any time, taking on a sudden acceleration without apparent cause, even after many years' duration.

Never is there spontaneous involution, and rarely do the tumours become ulcerated. Malignancy does sometimes supervene.

As to the histology, Brooke [3] says:—

"At the first glance, under a low power, some of the sections closely resembled an adenoma of the sweat coils, and a careful examination with a high power was required to differentiate between the new element and the exaggerated coils of a sweat gland. But however tempting it may seem to regard these tracts as allied in some way to sweat ducts or glands, a careful examination shows that they really have not at all an identical formation, and that the tube-like form which they assume is only apparent. I have never been able to find a single instance of a true lumen in either the tracts or the masses. Two long parallel rows of small palisade cells, almost meeting at their base, seem frequently to show a clear space between them, as if they really formed the wall of a tube, but a high power has shown invariably the existence of one or more rows of elongated cells of much lighter colour filling up the supposed cavity, and only when one of these cells which has become colloid, as is frequently the case, is seen in cross section, is the illusion of a lumen at all troublesome.

"[The illustration] shows their rope-like tracts, or finger-like prolongations of epithelium, cut across at all angles, and evidently representing the section of an intricately convoluted mass. Lying in these tracts, but more generally in the masses, were cysts of circular or oval shape, filled with either purely colloid matter, or partly with colloid and partly with concentric layers of apparently horned epithelium (epithelial pearls). In some sections this cyst formation was elementary. In others it constituted the most striking features of the picture. The connective tissue which surrounds the epithelial growth is not the ordinary normal tissue of the corium, but a mass of much finer texture, and is, as it were, moulded round the contour of the growth to form a dense capsule. Its density and thickness both indicate the age and slowness of the rate of growth of the tumours."

Brooke found the tumour arose from the basal epithelium of the surface and of the follicles, and he epitomized its characteristics in his designation of epithelioma adenoides cysticum. Fordyce's [9] account substantially confirms Brooke's findings.

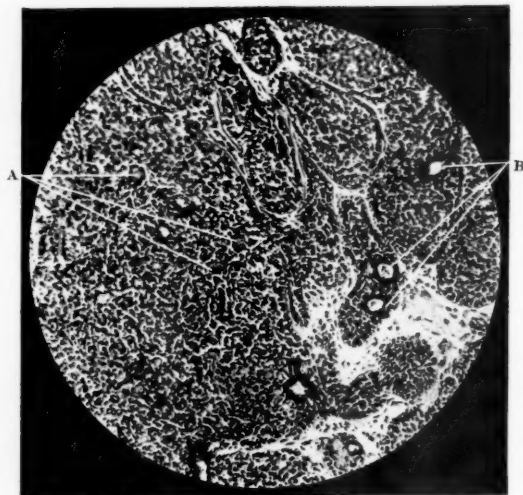


FIG. 6.—Case III. Section of scalp tumour showing "hyaline bodies" (A) and small cysts (B).

This adenoid character of the cell masses was responsible for Perry's [17] diagnosis of "adenoma of the sweat glands" and it, together with the inclusion of sebaceous gland substance, led to Balzer's and Ménétrier's [2] title of "Adenoma of the Sebaceous Glands," though, as Unna points out, their fig. 3 distinctly shows that what the authors had described as sebaceous cells were in reality cells which had undergone hyaline degeneration, or were forming epithelial pearls. Unna [22] further states that the constant palisade form of the peripheral epithelia, as well as the firm connexion of these epithelia in general, even in their most reticular structure, indicates—though Brooke and Fordyce did not note it—a developed system of epithelial fibrillation. He therefore suggests that the growth is a true acanthoma, but Wolters prefers the term "epithelioma" to that of "acanthoma," since the latter definitely

means proliferation of prickle cells, where prickle-cell structure is maintained, which cannot be affirmed of the condition under discussion.

Jarisch [12] traced the origin of the tumours from the basal cells of hair follicles.

Pick [18], on the other hand, found a marked proliferation at the periphery of the sebaceous acini, forming epithelial tumours similar in appearance to those of Brooke's, and suggested "adeno-epithelioma" as the most suitable name.

Csillag's [5] illustrations show the growth arising both from the basal cells of the surface epithelium and of the hair follicles.

Now it is essential in investigating this subject to examine very many tumours, and tumours from all situations. We have found, clinically, that the growths vary considerably in different situations, and so, too, this variation is reflected in our histological pictures (of which I will now throw on the screen a few slides by way of illustration). Neither Brooke nor Fordyce appear to have examined growths from the scalp, or they would have found here a rather different picture. As I have already indicated, the milium-like bodies are not apparent in this situation, and consequently sections show only very small cysts (fig. 6). The adenoid masses are densely packed and surrounded by a clear hyaline stratum, whilst numerous hyaline bodies (or rudimentary cysts) are present in the alveoli, and, according to Dubreuilh and Auché [8], these bodies are not present in basal-celled carcinoma. These authors also confirm the findings of Brooke [3] and Fordyce [9], showing that this hyaline degeneration is evident in the masses soon after their origin from the basal epithelium, but they erroneously describe as blood-vessels within the alveolar masses what are in reality early cysts.

With regard to the formation of the cysts, Brooke [3] says:—

"Epithelial cells become large and translucent, the cells around them take on a like action, and a little focus of degenerated cells is thus produced. Owing apparently to the centrifugal and lateral pressure, the neighbouring cells assume a cubical shape and form a kind of wall round the central mass. The cells constituting the original focus increase in size, lose their clear contours, form translucent clumps in which the nucleus is very faintly stained, and later disappears. As the mass grows the cells at the periphery become more and more flattened and condensed, until they at length consolidate into a solid cyst wall. The contents vary; in some of the cavities they are completely colloid, in others they are composed of concentric layers of flattened horny cells round a colloid centre; or again part of the contents may consist of a colloid mass and part of horny layers, but I have not found one cyst in which the whole of the contents was made up of horny cells." (Fordyce differs on this point, and states that some of the cysts were entirely filled with horny cells.) "The presence of colloid matter seems to be an essential factor in the origin of the cysts, even if the later additions to its circumference undergo the more normal transformation into corneous tissue. It is only when the colloid mass reaches a certain size and thus produces a corresponding amount of outward pressure that a true cyst is formed."

Brooke also points out that this degeneration is not confined to the older tumours, but seems to be an essential feature of the growth from its beginning. Large cysts may be found in small tumours whilst the largest growth may be free from any except those of the smallest size.

The cyst grows at the expense of the neighbouring epithelium, and may absorb the cells so completely as to give the appearance of being isolated in the connective tissue, but in the other section the trace of epithelium connecting it with the body of the growth, and in which it has originally

formed, may be found intact. In consequence of the apparent snaring off of the cyst Csillag [5] has suggested that there are two kinds of cysts formed, one which results from the colloid degeneration of the epithelial masses and the other from the snaring off of the follicle and the production of a true sebaceous retention cyst, the latter corresponding to the small tumours which resemble milium.

The slide (fig. 7) now shown demonstrates fairly clearly the formation of the cyst as described by Brooke. Csillag's view of a dual origin of the cyst cannot, I think, be maintained.

I fully agree with Brooke's explanation, except that I would substitute *hyaline* for "colloid" and *formation* for "degeneration." The slide next shown portrays very clearly the formation of hyaline bodies and the further development from them of small cysts which Dubreuilh and Auché refer to as blood-



FIG. 7.—Case IV. Section showing the evolution of the cyst.

vessels, but one finds the blood-vessels in the septa between the alveoli, and not in the cell masses. These hyaline bodies are not present in all sections. They are not so evident in the tumours on the face and trunk, nor are they always to be found in scalp tumours. The hyaline formation in the connective tissue is far more constant in scalp tumours, but is found also in growths from other situations.

Dr. Gilchrist, who saw these sections lately, noted the similarity of these cysts to sweat ducts, and suggested that perhaps they might be such, accidentally included in the new growth, but on further investigation I am convinced that they are not connected with the sweat apparatus, but that they result from an attempt on the part of the cell masses to produce hair follicles.

Other slides show, too, very clearly, the attempt at the formation of sebaceous glands by the adenoid masses; so that we have an attempt on the

part of the new growth to reproduce the pilo-sebaceous apparatus, and this hypothesis is, I think, further supported when we consider the rôle of the pigment found in not a few of these tumours. A casual clinical inspection, it is true, will find black "dots" in but a few of the tumours, though many of the growths present a slight brownish or bluish tinge, and this colouring is due to the presence of a small amount of pigment in the cell masses or in the stroma *beneath* the epidermis (fig. 8). A careful histological examination will however reveal some pigment in very many unsuspected instances. This pigment is elaborated by the epithelium, and its manufacture completes the picture of an attempt on the part of the cell masses to produce a hair follicle with its attendant gland. The pigment appears to be quite passive and does not induce malignancy.

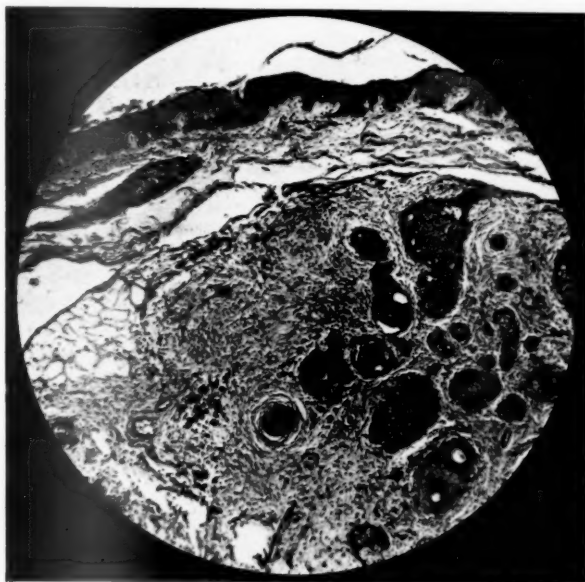


FIG. 8.—Case III. Section of a dorsal tumour showing pigment in the alveoli and in the stroma.

Three things are, therefore, essential in a histological picture of this condition: (a) basal epithelial cells; (b) massed in adenoid formation; and (c) encapsuled by firm connective tissue.

There is no time to discuss differential diagnosis in full detail, but I would specially advocate the separation of this condition from the two other well-established varieties of so-called benign cystic epitheliomata, namely, tricho-epithelioma of the lids and syringoma of the trunk. Our modern text-books do not emphasize the distinction as did the older ones. I am aware that the fusion of what I consider distinct clinical and histological entities, into one large group of benign cystic epithelioma, reflects a large body of present-day dermatological opinion, and this opinion is largely based on the fact that these tumours all



have their origin in the basal layers of the epidermis, and that some few cases have been recorded which are very difficult of classification. I submit that there is as much justification for the separation of these benign epithelial tumours as there is for separating the basal-celled from the prickled-celled carcinoma; for Darier has recently emphasized the fact that we have a mixed variety—the baso-spinal epithelioma (and I would add, too, a spino-basal epithelioma). I have thrown on the screen many illustrations of Brooke's disease which are uniform in type. In the so-called tricho-epithelioma (or cystic naevi) of the lids the lesions are usually confined to the eyelids, though they may be found between and in the neighbourhood of the brows. The condition is not rare. I see a case on the average once a month. A family history of heredity is rare. Histologically the cystic element is very marked, and instead of adenoid alveoli we have epithelial strands running parallel with or at right angles to the surface. Many of these strands present a duct-like appearance.

In the syringoma, or misnamed "eruptive" hydradenoma, the distinction is even more marked. The lesions are as a rule more deeply embedded in the true skin, are firmer and do not attain the size that it is now known many of the lesions do in Brooke's disease. These nodular tumours are usually limited to the trunk. Unna [22] says:—

"The syringoma tends to tubular formation; is long independent of the surface epithelium; has never any connexion with the hair follicle; and never shows lobulated or branched masses, but always the duct form; or it forms isolated cysts; induces no displacing pressure on the normal elements of the cutis and is restricted to the central parts. We never find the epithelial cylinders of the syringoma encapsuled by a special connective tissue sheath and there is no epithelial fibrillation nor prickles in the cells."

The only thing analogous to both tumours is the formation of colloid or hyaline cysts.

Crocker and Hartzell are strong advocates for the separation of these conditions, and Dr. Adamson—our President—puts the case very clearly in his paper of 1914.

I think it is impossible to differentiate clinically with certainty between a solitary lesion of epithelioma adenoides cysticum and a non-pigmented or pigmented mole. A correct diagnosis can only be made after examining a section, and it would be equally difficult without a section to differentiate a simple ulcerated lesion from a mole in which a rodent ulcer or epithelioma had developed. The differentiation, clinically, of a small solitary lesion from a non-ulcerated rodent ulcer is not easy, though in the latter a real and not an apparent depression or umbilication is often present and on palpation there is less resistance.

With regard to the differentiation of the multiple variety from multiple rodents I think there should be little difficulty. Dr. Adamson, I am pleased to learn, no longer holds to his differentiation enunciated some years ago. The main points are: (a) The uniformity of the lesions, the rarity of their ulceration; (b) the absence of atrophic scarring and the hereditary transmission of the benign tumours as compared with the multiformity of the lesions; (c) their tendency to ulceration and to atrophy and the absence of the hereditary factor in multiple rodents. There is just the possibility that some cases which have been shown before this Section may have been instances of mixed benign and malignant tumours [7].

With regard to their histological differentiation I cannot do better than quote the conclusions of Dr. Adamson, who says:—

"In both diseases the lesion is made up of an epithelial growth derived from the basal layer of the epidermis and from that of the hair follicles. The growth is in the form of cell masses with a marginal palisade layer and central oval cells. There is a tendency to cyst formation in both colloid cysts and epidermal cysts. In both the epithelial masses have new formed encapsulating fibrous tissue. Here, however, the resemblance ceases and we find that while the lesion of Brooke's disease is sharply circumscribed by a highly organized fibrous tissue, there is in the rodent ulcer a more highly cellular and therefore more actively growing fibrous tissue element, a plasma-cell exudation at the advancing margin and out-lying groups of epithelial cells invading the tissue beyond the main growth."

When however the tumours are situated on the scalp and back there should be no difficulty at all.

There is a condition for which a case of moderate distribution might be mistaken and that is the so-called adenoma sebaceum, especially of the non-telangiectatic variety. Heidingsfeld expresses some doubt as to whether a patient whose picture he presented to Pusey and which appears in Pusey's text-book may not have been a case of epithelioma adenoides cysticum.

Lastly, there is the differentiation of the cases which present multiple growths on the scalp only. I quite endorse Adamson's statement that "the majority of cases so-called *endothelioma capitis* belong to the group of cases which are known as epithelioma adenoides cysticum (Brooke)," but I do not agree that *all* the cases he cites belong to this group for some I consider undoubted cases of cylindroma. These latter tumours may be solitary or multiple on the scalp and neighbouring parts. They are not usually seen before middle age and may remain dormant for some years, but later may become ulcerated and invade the deeper tissues. They are of equal malignancy with rodent ulcer.

Histologically the tumour is made up of (i) cylinders of cells for the most part anastomosing to form trabeculae and giving the appearance of glandular structures; (ii) of cellular masses which are really actively growing cylinders in which are formed cavities the result of degeneration. The most characteristic feature, however, is the mucoid degeneration of connective tissue which according to Malassez grows into these masses but which, as Nicolau has demonstrated, becomes imprisoned in the network of trabeculae formed by the cylindrical growth. The histological picture is quite distinct from that of epithelioma adenoides cysticum. The myxomatous degeneration is seen in the earliest tumour and is quite characteristic. Dalous [6] carefully compared sections of cylindroma with sections from Dubreuilh and Auché's case of benign epithelioma.

#### TREATMENT.

For solitary lesions "excision" is the best. For the multiple variety electrolysis or excision are indicated when the lesions are not very numerous. In the more extravagant cases X-rays clear up the tumours but the milia remain and require expressing.

Of the forty-three recorded cases we have just reviewed, thirty were in females and thirteen in males; thirty-five showed multiple lesions and in eight solitary tumours alone were evident. In twenty-eight there was a positive family history, while in fifteen the family history was negative. Only in one instance of a patient presenting a solitary lesion was the family history positive and in this case (boy, aged 12) the tumour was probably the forerunner of others. We have noted the difficulty, if not the impossibility, of making a correct diagnosis of the solitary tumours by clinical means alone for they

present the appearance of the common so-called "epithelial" mole. All authorities agree that these tumours, apparently of embryonic origin, belong to the large group of nævi; and though I hesitate to add to the list of titles, already too large for enumeration, I cannot conclude without suggesting that instead of benign epithelioma or acanthoma, we should adopt some such title as "nævus follicularis" or "follicular nævi of the skin."

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## Section of Dermatology.

President—Dr. H. G. ADAMSON.

### Case for Diagnosis.

By W. JENKINS OLIVER, B.M.

GIRL, aged 7, in apparently good general health. The skin lesions are confined to the back and right flank and consist of five round and irregularly quadrilateral erythematous macules, some of which show fine scaling. The lesion on the back of the right shoulder differs from the others in being darker in colour, of a slightly bluish tint with a white shade in the centre. Over this, the oldest macule, there is, on palpation, the suggestion of a lack of suppleness of the skin which is not apparent over the other lesions. The duration of the various lesions has been: (1) back of right shoulder, eighteen months; (2) small round patch on left side of spinal column over scapular region, fifteen months; (3) one round and one large irregularly shaped area extending on to the right flank, about ten months; and (4) irregular quadrilateral lesion over right sacro-iliac region, four months. Since their first observation the spots, according to the mother's statement, have appeared to become larger, especially the one on the right flank. The oldest lesion on the right scapula has always been different from the others, with its white centre. There is no history of trauma, nor of drug-taking. Neither the patient nor the parents have been abroad, and there is no apparent alteration of sensibility over any of the macular patches, while the child complains only of some occasional irritation about them. I show this case as one for diagnosis, putting forward the tentative suggestion of localized sclerodermia, in which case the purely erythematous macules show how slow may be the evolution of the sclerodermatous condition.

### DISCUSSION.

Dr. H. G. ADAMSON (President) said that this type of sclerodermia, with one or more oval patches generally on the back, was not uncommon in children. They were sometimes associated with erythematous rashes or urticaria, and more rarely with fluid in the joints—evidence perhaps of a general toxæmia. The erythematous patches in Dr. Oliver's case were, he thought, an early stage of the sclerodermia; they had the peculiar lilac colour of the spreading margin of a sclerodermic patch and already showed sclerodermic changes to a slight degree. The prognosis in this type of case he regarded as good. He had not found that any form of treatment had any good result, but the sclerodermia patches often disappeared spontaneously in course of time.

Dr. GRAHAM LITTLE said that he showed a similar case in a child, aged 9. The condition was on the back, and there was a very distinct history of acute onset. The eruption appeared during the time she was attending the hospital, a patch the size of a half-crown developing in three weeks, and there was an erythematous blush, as in this case. Three of the patches underwent spontaneous involution in six months, leaving no trace whatever.

Dr. S. E. DORE said he thought the erythema stage of sclerodermia was not sufficiently recognized. The patient in a case which was under his care gave a history of recurrent attacks of "eczema," but they were attacks of erythema preceding the sclerodermia. The sclerodermic patches were not only preceded by a margin of erythema before they spread, but the patient had localized patches of erythema, which eventually became sclerodermatous patches.

### Leishmaniasis of the Skin resembling Lupus Vulgaris.<sup>1</sup>

By J. B. CHRISTOPHERSON, C.B.E., M.D.

THE case which I bring to-night is, in my opinion, one of an hitherto unclassified form of leishmaniasis of the skin, resembling lupus vulgaris. It was shown on July 20 this year (1922). Of the speakers on that occasion I think Dr. Whitfield alone agreed with the diagnosis of leishmaniasis. The patient, a staff nurse, was bitten on the cheek by a biting fly whilst doing hospital work with the army at Baghdad in September, 1920, and a small single sore formed. Being invalided for tachycardia, she was sent to Netley Military Hospital, where the sore was investigated, Leishman-Donovan bodies found, and the lesion was diagnosed as Baghdad or Oriental sore. It gradually improved under treatment with methylene-blue ointment and antimony ointment (no intravenous injections were given); the X-rays were applied once. It never completely disappeared, and in June, 1922 (a year after her discharge from Netley apparently cured), she came before a medical board and was sent to the tropical diseases clinic.

On June 9 of this year, when she began to receive intravenous injections of antimony tartrate, there were about twenty-five vesicular lumps of varying sizes, which, when taken between finger and thumb, looked yellow and round, and were soft. The skin over the lumps was not ulcerated or broken. The nodules, when punctured, were found to be hollow, and were filled with an apple-jelly-like content, similar to what is found in lupus vulgaris. These nodules were grouped around the scar of the original Oriental sore. To-day the lumps have vanished, the area of skin occupied by the vesicular nodules is level with the surrounding skin, and the nodules themselves are not much more than mere stainings.

I do not think that lupus vulgaris would have shown the improvement this condition has shown in three months under any circumstances whatever. The patient is practically well four months after treatment with intravenous sodium antimonium tartrate commenced; given in single doses, commencing with  $\frac{1}{2}$  gr., increasing to 2 gr. per dose, a course of 30 gr. was administered at first, and, after an interval of two months, a subsidiary course of 12 gr. has been given.

I may say that Leishman-Donovan bodies were found in the original single Oriental sore, one and a half years ago, which apparently healed, and degenerated Leishman-Donovan bodies were found in the nodular sore which I describe to-night. Also "blue bodies" were found abundantly in the smears. Blue bodies are round bodies of variable size which stain a uniform blue colour with methylene-blue, apparently structureless; they are found invariably in smears and sections of leishmaniasis specimens. I am the more inclined to the diagnosis of leishmaniasis because this is not the only case of the kind

<sup>1</sup> For the illustration of this case see *Proceedings*, 1922, xvi (Sect. Derm.), p. 9 (fig. 1).

I have seen. I have another case, similar to this one, under observation; the patient is also a nurse, and also from Baghdad: in her case, too, the nodular sore occurred on the face; and another point of similarity between the two cases is that the sore on the face, appearing after the original sore, on the arm in the second case, had been apparently cured for some time.

Now the clinical story in these two cases is suggestive of an imperfectly cured or residual condition, or perhaps a modified form of leishmaniasis, the variation of which from type is due to treatment. In neither of these cases was the original sore treated with intravenous injections of antimony tartrate. In both cases, the Leishman-Donovan bodies were either absent or difficult to demonstrate, but there is no doubt about the case I show to-night being very susceptible to the specific remedy of leishmaniasis—intravenous injections of antimony tartrate. There is no doubt also about the lesion being very similar in appearance to lupus vulgaris, and I describe it as leishmaniasis of the skin assuming a lupoid form.

#### DISCUSSION.

Dr. J. M. H. MACLEOD said that he thought the appearance of the lesion suggested that the leishmaniasis might have become secondarily infected with tubercle, as the lesion was indistinguishable from lupus in its appearance. He would have liked to have seen a section, as it might have assisted in solving a problem.

Dr. ARTHUR WHITFIELD said he had examined sections from a case of what afterwards proved to be leishmaniasis, and he found himself unable to diagnose, under the microscope, the non-ulcerated leishmaniasis from one type of lupus. He had, indeed, made the diagnosis of lupus in a case which afterwards was shown to be leishmaniasis. It would be of help if sodium antimonium tartrate were tried by injection in cases of undoubted tubercular lupus, as this might show that the beneficial result obtained in this case could not be obtained in lupus. He believed antimony had been used for the condition by giving it freely per os many years ago, before Koch's first tuberculin came out, because it was stated a reaction could be caused by giving large doses of arsenic or of antimony.

Mr. McDONAGH said he had seen three cases of leishmaniasis in which after the disappearance of the primary lesion papules appeared in the periphery. These papules were the result of direct spread along the lymphatics from the original focus, as was proved by serial sections. Unless the phases of the life-cycle of the organism were demonstrable in a section it was impossible to differentiate between leishmaniasis, tubercle, syphilis, &c., as these diseases were capable of producing the same histological picture. Development of papules by direct extension from the primary lesion which was so common in syphilis was not at all rare in the condition under discussion.<sup>1</sup>

Dr. H. C. SEMON said he had a very resistant case of lupus vulgaris of the nose, which he had had treated by Finsen light. It was not very successful, and the patient came back twice with recurrence after apparent cure. Dr. Christopherson and he then tried a course of antimony injections by way of comparing its effects with those achieved in the nursing sister's case. He did not think there had been any improvement, and certainly not to the degree shown in the case presented by Dr. Christopherson.

Dr. CHRISTOPHERSON (in reply) said that this case was tried with the intravenous injection of sodium antimonium tartrate, with the idea it possibly might be lupus vulgaris. The question of making a section from the lesion was put to the patient, but as the condition was obviously getting well, it did not seem quite fair to do a biopsy, especially as the site of the sore was a conspicuous one. Animal injections were not thought of until too late. Typical Leishman-Donovan bodies had not been seen in this

<sup>1</sup> See *Brit. Journ. Derm.*, 1915, xxvii, p. 91; 1921, xxxiii, p. 182.

case since June—five months—when the warty appearance occurred, but this was not altogether against the diagnosis of leishmaniasis; in a number of other diseases one was not always able to find the causal organism. In the present case "blue bodies" had been found repeatedly, and these he looked upon as almost diagnostic of leishmaniasis. Some were degenerated Leishman-Donovan bodies, some no doubt the cytoplasm of tissue cells broken up by the Leishman-Donovan bodies.

### Case of Folliculitis Decalvans.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a lady, aged 57. I think this is a case of the pseudo-pelade of Brocq, for although there is very little evidence of active folliculitis, there is a peri-follicular ring round several of the hairs, giving the coarse "orange rind" aspect of the skin which is often found in Brocq's pseudo-pelade. Whether it is desirable to make this type a separate entity from folliculitis decalvans is a matter of opinion. In one of our discussions, Dr Pringle pressed for the non-separation of cases of pseudo-pelade from the class of folliculitis decalvans; unless we reserve the former name for differentiation of a group of these cases showing definite atrophy, but without a very active suppurative stage. I shall be glad if it can be shown that this is a more hopeful form of alopecia. It is still extending in this patient. She has had thinning of the hair for a number of years, but it was only distinctly noticeable from last July. She has also a long history of what is probably colitis, dating from a visit to Buxton eleven years ago, when there was, at that watering place, an epidemic of choleraic diarrhoea, probably from a food poisoning infection. She has ever since had an inflammatory condition of her intestinal mucosa, and her custom has been to have several loose motions per day. That part of her trouble, however, has now been materially improved by adopting Dr. Guelpa's treatment. I do not know whether the colitis is related to the skin condition.

#### DISCUSSION.

Dr. S. E. DORE said he did not contest the diagnosis in this case, but it did not resemble any case of pseudo-pelade he had seen; it was much more diffuse. Generally the transition between the cicatricial area and the normal hair was more abrupt. He had now under his care two patients with lupus erythematosus affecting the scalp without any lesions on the face or body, and it was possible that the case under consideration might be an instance of this disease.

Dr. DOUGLAS HEATH mentioned a type rarer than the diffuse variety, in which there were patches with a swelling round the affected hairs, and the hairs broke off short, as in alopecia areata. The swelling was marked, and there was itching and irritation; the hairs fell out, and did not re-grow. The affected hairs were very œdenatous; when a hair was withdrawn the sheaths were very swollen. He had not found any organism in them, though he had made many cultures. He did not know whether that condition should be grouped with the disease now being discussed.



## Two Cases of Favus of Smooth Skin.

By E. G. GRAHAM LITTLE, M.D.

THE patients are a boy aged 6 and a girl aged 8; the former has a patch of favus on the chin, the latter one on the right ear. Both have become very much better with the use of boracic ointment which I prescribed as a placebo. The ear lesion has almost entirely cleared up. The favus fungus was very obviously present in both lesions. I have not seen a case of favus for five or six years, and the disease is becoming increasingly rare. It is difficult to know where this is likely to have come from. The house is not infested with mice. The children are resident in London, and have been for the past three years, having originally come from India. There was no infection before they left India, and the whole of the symptoms have appeared during the last six weeks.

### DISCUSSION.

Dr. A. M. H. GRAY said that he had been talking that day to Mr. Foulerton, of the Public Health Department, University College, who said he had recently seen a good deal of favus in mice. He (Dr. Gray) thought there was a certain amount of scalp favus about; there had been two cases at his clinic in the last few months, but the patients did not show the classical signs of the favus cups.

Dr. H. G. ADAMSON (President) said that unless scutula were present it was not possible to distinguish favus of the glabrous skin from *tinea circinata* without making a culture.

## Case of Darier's Disease.

By H. W. BARBER, M.B.

PATIENT, a male, M. C., aged 11; an orphan and illegitimate child. His mother had another child, a girl, by her husband; this girl is now aged 21, and is alive and normal. The patient's father was a single man and is not known to have had other children. Neither parent had any chronic skin disease. The eruption began more than six years ago, i.e., before he was 5 years old. It appeared first on the backs of the hands and on the extensor surfaces of the legs. The face has become affected only recently. The eruption involves chiefly the backs of the hands and wrists, the forehead, temples and sides of the nose, the neck, the legs below the knees, the groins and axillæ. On the forehead crusting has occurred, but the scalp is only slightly affected. The lesions on the hands and wrists resemble flat warts, and on the face the greasy appearance and follicular involvement are well seen.

Dr. J. M. H. MACLEOD said he had had a very extensive case of Darier's disease under his care for a long time; the legs were badly affected. Owing to the keratinization being so imperfect the skin of the leg became septic and eczematized. This healed up on X-ray exposure, but no permanent effect was produced by the rays on the Darier's disease.

**Squamous Carcinoma of Face in a Woman aged 24.**

By W. J. O'DONOVAN, M.D.

M. B., A SINGLE woman, aged 24, first attended Dr. J. H. Sequeira's out-patients department at the London Hospital on January 23, 1920. She then presented a painless longitudinal ulcer over the right lower jaw of seven years' duration. There was a similar smaller area below her left ear. No X-rays had been applied; there was a narrow area of white scarring around the ulcer. Twenty treatments with Finsen light and the application of ointments had not produced any good results by the end of 1921. Four applications of X-rays, half to a whole Sabouraud pastille doses, were also of no value, but as the patient had moved far out into the country her visits were now infrequent. By October, 1921, there was still an oval shallow ulcer on the skin over the right lower jaw, with a white highly vascularized livid margin. Thick crusting up of the ulcerated surface was a marked feature at every attendance. In July, 1922, the ulcer had a "nodular-rolled edge." To-day she presents an oval pink ulcer over the right lower jaw, 12 by 7 cm., with a narrow raised rounded pale margin traversed by fine vessels. Below the left ear there is an irregularly shaped rounded skin lesion, pink, slightly sunken, having a definitely raised palpable margin with a smooth non-ulcerated surface. There is no adenitis. The lesions are apparently no larger than when first seen in 1920. On August 25 a portion of the edge was removed for microscopic examination.

## REPORT BY PROFESSOR H. M. TURNBULL, M.D.

The specimen is a portion of skin, measuring 6 mm. long by 1.5 mm. broad by 2 mm. deep, in formaldehyde solution. On the surface is a brown area (5 mm. long); the remainder of the surface is finely wrinkled.

*Microscopic Examination.*—The skin contains sebaceous glands, hair roots and hair follicles. In the centre of the specimen there is a depression in the epidermal surface. Broad processes of epidermis extend downwards deep into the dermis beneath this. Round these processes there is a conspicuous infiltration of the dermis with lymphocytes, occasional plasma cells and a very few eosinophil leucocytes. Here are very many distorted cells, which are represented by fusiform or thread-like pyknotic nuclei. Similarly distorted cells are present in other portions of the dermis. The papillary zone of the epidermis shows a focal area of oedematous rarefaction. Within the enlarged and elongated epidermal processes are three rounded masses of horn containing a few nuclear remnants. Similar masses of horn are seen in the mouths of hair follicles in other parts of the section. In the deeper portions of the processes the cells are slightly swollen, and prickle-borders are absent. Prickle-borders are distinct in the greater part of the remainder of the epidermis, but not throughout. In other respects the cells are not atypical. The outer margins of the deeper portions of the processes are irregular and very poorly defined. They are not limited by the distinct layer of basal cells which is present in the remainder of the section. The lack of definition of the processes is in part due to the margins being crossed by many of the fusiform and thread-like distorted nuclei. The elongated and downward processes of the epidermis cannot be explained by wrinkling of the skin and tangential section. They appear to be the result of overgrowth of the epidermis. The

three included masses of horn suggest that the overgrowth of one process follows a tortuous hair follicle. The growth is associated with infiltration of the adjacent dermis. The absence of the basal layer, and the irregular boundary and poor definition of the processes give evidence that the overgrowth is atypical. The lack of definition is largely due to the distortion of cells, particularly infiltrating cells. The cellular distortion and the focal oedema are doubtless due to the injection of anæsthetic. Allowing for this artefact, I consider that the abnormalities in the margins of the processes make it necessary to regard the overgrowth as carcinomatous, or potentially carcinomatous.

#### DISCUSSION.

Dr. A. WHITFIELD said he did not think the section taken had gone deep enough, nor was it sufficiently extensive, to enable a sound opinion to be formed. It was a chronic inflammatory trouble, and there was an immense amount of small-celled infiltration beneath; all the tissue concerned consisted of small-celled infiltration. There was also folding, and a biting off of bits of epithelium. A deeper section might show something more interesting below the band of small-celled infiltration.

Dr. H. G. ADAMSON (President) said that the case was of great interest, in view of the instances of superficial rodent seen by the Members recently. He thought this case was, clinically, exactly like those cases. There was a rolled edge, and slightly crusted or scaly centre. The extraordinary feature was that the section showed a squamous-celled growth instead of the usual basal-celled growth, such as had been described in all of the previously described cases with the same clinical type of lesion. He suggested that Dr. O'Donovan should make further microscopical sections to see whether the lesion showed a basal-celled growth in another part.

#### Case of Adenoma Sebaceum.

By H. C. SEMON, M.D.

PATIENT, a girl, aged 13, had developed the soft, closely aggregated, raised yellowish-pink waxy lesions in the nasolabial grooves and symmetrically on the cheeks, when 5 years old. Small telangiectatic stigmata were also to be seen, and there was a flat warty mole in the left frontal region.

To complete the picture of the Pringle type, the child was very poorly developed mentally, could remember nothing of what she was taught at a special school, and had suffered frequently from epileptiform seizures, from an early age. An uncle on the father's side is said to have died in a fit, after many attacks. Microscopic sections showed embryonic hair follicles, sebaceous and sweat glands. The Weigert stain proved the sparsity of elastic fibres, and the whole histological appearance strongly suggested the nævoid origin of the disfigurement.

It was proposed to destroy the lesions individually, in a number of sessions, by means of the electric cautery.

#### Case of Epidermolysis Bullosa.

By W. F. R. CASTLE, M.B.

THIS mother and her baby have epidermolysis bullosa. The disease can be traced through four generations, the great-grandfather, grandfather, mother and child being affected. The mother is one of eight children, all of



## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case of Bullous Eruption.

By J. H. SEQUEIRA, M.D.

THIS patient is a boy, aged  $3\frac{1}{2}$ ; there is another child in the family, aged 13 months. The family history is unimportant. The child's general health is good; there is no evidence of visceral disease, but for the last six months there has been an eruption of bullæ. These come out singly and in groups, and they vary in size from that of a split pea to that of half a cob nut. They contain perfectly clear serum, and the base, at the onset, is free from erythema. Practically the whole surface is covered with groups of blebs. There is some irritation at night, but the child takes its food well. The temperature is quite normal. There is slight eosinophilia of the blood, 10 per cent., but no eosinophils in the serum. At first the blebs are free from organisms, so far as cultures show: later, however, they apparently develop *Staphylococcus epidermidis albus*. Following the example of the late Dr. Colcott Fox, I have entitled the case one of "bullous eruption."

The question whether this is pemphigus or dermatitis herpetiformis is purely one of nomenclature, or of what one understands by those terms. Taking the term "pemphigus" to mean a bullous eruption where the blebs come out on a clear skin and where the disease is not essentially polymorphous, this should be classed as pemphigus. From the cases seen in the past, I think this case will do well, and I think most here agree that it is a condition which usually reacts well to arsenic.

Dr. GRAHAM LITTLE said this case revived an old discussion. He had always urged that the division of bullous eruptions into two classes, "pemphigus" and "dermatitis herpetiformis," was premature, and that the group should be recast. He did not see why the name "dermatitis herpetiformis" should not be applied to this case, as it seemed to fulfil many of the criteria of that condition. He had looked through his own cases, and found he seldom recorded having seen pemphigus, whereas Dr. Radcliffe Crocker appeared as seldom to have seen dermatitis herpetiformis; though no doubt both he and Crocker had seen the same types of cases and labelled them differently. Therefore Dr. Sequeira acted wisely in calling this a bullous eruption—it was admittedly either dermatitis herpetiformis or pemphigus.

**Case for Diagnosis.**

By H. G. ADAMSON, M.D. (President).

THIS young woman was referred to me some months ago by Dr. G. A. Hooton, of Durban, South Africa. She lives on a farm in a village about seventy miles from Durban, and before this had worked for some years in a general store where she served both Europeans and natives. During the past five years there have appeared upon her face and other parts deep-red mahogany-coloured plaques, the nature of which is doubtful. Dr. Hooton was inclined to regard them as lupus erythematosus or lupus vulgaris: others who have seen them have diagnosed discoid, superficial rodent ulcer or basal-cell epithelioma of the type of which we have seen several examples lately. When I first saw the patient I hesitated as to the diagnosis of lupus vulgaris to which the plaques certainly bore a close resemblance and came to the conclusion that they were the lesions of leprosy of the macular variety, a diagnosis which I am interested to find has also suggested itself to Dr. Castellani, who has seen the patient this afternoon.

The distribution of the lesions is as follows: Eight on the face, viz., one at the outer third of each eyebrow, three on the nose, one on the upper lip, one over the lower jaw on left side and one in front of the left ear; three on the scalp; one on the front of the neck, and one on the left forearm. They vary in size from  $\frac{1}{4}$  in. to  $1\frac{1}{2}$  in. in diameter. They are irregularly rounded, very sharply margined, with a smooth surface not raised above the level of the skin, and semi-translucent aspect not unlike that of a superficial lupus infiltration. They differ however from lupus patches in the superficial infiltration being uniform and not nodular and in their curious deep mahogany colour. Some of the patches, notably that one in front of the left ear, show pale leucodermic-like areas as though part of the infiltration had disappeared, leaving a fine atrophy. One of the lesions, that on the upper lip, has a slightly raised margin which gives it somewhat the appearance of a rodent ulcer, and on very close inspection the same narrow raised margin can be seen faintly indicated in the other lesions. The patches on the scalp show a central part where there has apparently been ulceration which has left scarring and destruction of the hair follicles. All the patches seem to be hypersensitive to the touch of a needle, compared with the surrounding skin. The patient has no other signs or definite symptoms of leprosy. She has complained for some months of feeling very tired and ill and of pains across the chest.

In order to aid the diagnosis, serum from a scraping of the surface of a lesion was examined for leprosy bacillus, but with negative result. A piece of the plaque on the forearm was removed for microscopical examination. It shows neither a basal-cell epithelioma nor a granuloma, but, in my opinion, the structure of a xanthoma. The epidermis is intact; in the corium from immediately beneath the epidermis to half-way towards the subcutaneous fat there is a very sharply circumscribed cell infiltration made up apparently entirely of so-called "epithelioid" cells, or oval cell nuclei, separated from one another by translucent finely granular masses which surround each cell or nucleus. Some of the "nuclei" are rounded, but these are probably oval nuclei in cross section and not lymphocytes. There are no outlying lymphocytic "muffs" such as one sees around the main cell infiltration of a granuloma, and outside the sharply margined infiltration the corium appears normal.

Dr. Barber, who has also made a microscopical preparation from another plaque, suggests that the appearances are those of a "sarcoid," but I am still inclined to regard them as those of a xanthoma in spite of the fact that the clinical features do not suggest a xanthoma. There is no sugar in the urine.

#### DISCUSSION.

Dr. A. WHITFIELD agreed that this was a very difficult case about which to be sure. He had been wondering whether it was possible always to diagnose leprosy correctly by the presence of bacilli in the skin in the anæsthetic cases. The bacillus was, he thought, found in the nerve trunk in them. The lesion exhibited under the microscope did not suggest an inflammatory condition; yet, in the absence of special staining, he did not feel convinced it was xanthoma. The nearest condition which the specimen suggested to him was neurofibromatosis, but that was so far removed from the clinical appearance that it must be a mere accidental resemblance.

Dr. F. PARKES WEBER said that five years ago he showed a patient as a case of some peculiar kind of xanthoma on the legs.<sup>1</sup> That patient had been a long time in England. After being exhibited, the case turned out to be one of leprosy.<sup>2</sup> It was interesting that in the present case Dr. Adamson's first thought was that the case was one of leprosy.

Dr. ARTHUR POWELL said he had seen a good deal of leprosy, especially in Bombay, where he was inspector of leprosy for a good many years, and visitor to an asylum of 450 beds, and this present case did not in the least suggest leprosy to him. In the nervous form of leprosy, it was almost the rule to fail to find the bacilli in the patches, but not uncommon to find them in the patient's nasal secretion when the nasal cavity showed no obvious lesion with the speculum.

Mr. J. E. R. McDONAGH said he had examined the section from this case and was struck by the sharply circumscribed character of the infiltration and the absence of polymorphonuclear leucocytes. The infiltration was made up, in the main, of endothelial cells and the predominating leucocyte was the lymphocyte. The capillaries showed a general thickening. The histological picture reminded him more of that produced by a slow growing coccidial protozoon rather than by anything else. It might be possible to discover the infecting agent if the section was stained with pyronin and methyl-green and examined with an oil-immersion lens.

### Case for Diagnosis.

By S. E. DORE, M.D.

PATIENT, a girl, aged 18, is shown for a peculiar condition of the right breast of five years' duration. There is no history of any preceding pathological condition of the breast, with the exception of an abscess in infancy, and there appears to have been no undue swelling or pressure dragging on the organ. The right breast is larger than the left, the swelling being more pronounced in the lower and inner quadrant. The skin in this region is puckered from atrophy and presents numerous small brownish infiltrations in its substance, grouped in a horizontal direction with minute slightly raised papular excrescences on the surface. There is no definite linear arrangement as would be expected in *lineæ atrophicæ*.

<sup>1</sup> *Proc. Roy. Soc. Med.*, 1917, x (Sect. Derm.), p. 164.

<sup>2</sup> *Ibid.*, 1920, xiii (Sect. Derm.), p. 12.



Dr. F. PARKES WEBER regarded this as a case of so-called linear atrophy (*striae atrophicæ*) of the skin, analogous to that not rarely seen about the shoulders, axillæ, or hips of rapidly growing adolescents. A determining local cause in the present case could probably be found in the manner of dressing, some garment or support keeping up a relative skin-tension below the large right breast. The exact nature of the right-sided mammary hypertrophy in the present case was of no special significance in regard to the cutaneous condition; which latter was due, as in all cases, to relative over-stretching of the skin, combined with active (normal or pathological) growth, or œdema or other swelling, of the parts beneath the cutis. The *striae atrophicæ* were the result of separation or cleavage of the cutis, as a method of adaptation; and some subjects were apparently specially predisposed to such *striae*.<sup>1</sup>

### Lymphangioma Circumscriptum of Tongue.

By Major G. PETIT, R.A.M.C.

THE patient, a young man, is convinced that this condition of the tongue only started in August, 1920, while he was in hospital with amœbic dysentery in Constantinople, and that it attained its present size in three months. At first the tongue was so sore that he had to take his food cold. Recently the soreness has returned. It was thought to be a sequel left from the dysentery. Lieutenant-Colonel Perry, of the Royal Army Medical College, diagnosed lymphangioma circumscriptum, and I have brought the case hoping to receive suggestions as to treatment.

Clinically the lesion involves most of the right half of the tongue, extending down to the floor of the mouth; the tip of the tongue is not involved. The growth is typical, consisting of thick-walled, pea-sized, opalescent frog-spawn-like vesicles, with small areas of telangiectasis scattered through it.

#### DISCUSSION.

Dr. A. M. H. GRAY said he thought there was only one thing to do in such a case as this, and that was to destroy the surface of the tongue by diathermy, or the cautery. Unless it was inflammatory, he did not think any X-ray or radium treatment would suffice. He had found extensive vascular nævi of the mucous membranes did very well when treated with liquid air.

Dr. H. G. ADAMSON (President) said he thought it questionable whether these lymphangiectases were always of nævoid character. If, as he believed, they were sometimes acquired inflammatory conditions there would be some prospect of doing good by means of X-ray treatment.

Dr. J. H. SEQUEIRA expressed his agreement with Dr. Gray's suggested treatment. He would apply the diathermy in patches, each time under a general anæsthetic, as he had seen great œdema follow extensive diathermy in the buccal cavity. He would work round the edges first.

Dr. GRAHAM LITTLE said he had a case, which he had referred for treatment to Mr. Cumberbatch, of a remarkably vascular nævoid growth, occupying the whole of the right cheek and the mucosa in the mouth. The patient was aged 17, and she had been under his observation twelve years. Pyorrhœa was definitely present, and the offensiveness of the mouth rendered some measure imperative. He first tried freezing, on the accessible parts, and for some time that seemed to be a success. But there was a re-growth. He then tried electrolysis, under anæsthesia, and for a short time this succeeded. Two months ago Dr. Cumberbatch applied diathermy, and now there was a recurrence as bad as ever.

Dr. S. E. DORE said he had seen many vascular nævi of the mucous membranes do very well under radium, and he thought it might be suitable in this case.

<sup>1</sup> Cf. F. Parkes Weber, "Unilateral *Striae Atrophicæ*," *Practitioner*, Lond., 1917, cix, p. 453.

Dr. SEMON agreed with Dr. Gray, and thought that there could hardly be a more positive indication for the use of electro-coagulation by diathermy, than an intra-buccal lymphangioma, such as was present on the tongue in this case. These tumours were notoriously susceptible to infection, and if such occurred here—as was not unlikely in the reactive stage after radiotherapy—he thought angina, or oedematous swelling of the larynx might occur, endanger the patient's life, and render a tracheotomy necessary. It had been found by laryngologists and others, that diathermy was of the greatest value in destroying malignant growths in the mouth and throat. Lymph spaces were sealed up by coagulation, there was no hæmorrhage, no sepsis, and practically no pain—and little or no reaction, as the eschar separated.

### **Case of Onychatrophia.**

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a girl, aged 10, began to develop these changes in the nails at the age of 5 years. One by one the affected fingers have lost their nails; apparently they have been absorbed, very much as in epidermolysis bullosa, but there is no other evidence of that disease here. The toes are quite normal.

#### **DISCUSSION.**

Dr. H. G. ADAMSON (President) said he had seen a similar nail condition associated with acquired syphilis. This child might have congenital syphilis, and he suggested that a Wassermann test be carried out.

Dr. F. PARKES WEBER suggested that this case was allied to cases of Raynaud's disease; and in Raynaud's disease one of the first things to find out was whether (by the Wassermann reaction or otherwise) any evidence of congenital or acquired syphilis could be obtained.

### **Section of Excised Pigmented Mole showing Early Malignancy.**

By E. G. GRAHAM LITTLE, M.D.

THE section was taken from a flat congenital pigmented mole on the shoulder of a medical student in my class. He showed it to me a week or two ago, and said it was causing him some irritation. As there was continuous friction from the braces, I suggested he should have it taken out. This was done. He is 24 years old. I have never seen a malignant change take place in a mole at anything like that age. The sections have been examined by Dr. E. H. Kettle, pathologist to St. Mary's Hospital, who regards them as showing definite early malignancy.

### **Pigmented Lesion for Diagnosis.**

By H. C. SEMON, M.D.

PATIENT is a woman, aged 59, whom I saw for the first time a week ago. She has had this rapidly growing discoid, pigmented and papillomatous neoplasm on her right cheek for about a year. Clinically it is freely movable on the subcutaneous tissues, there are no palpable glands, and the microscopic evidence is not conclusive of malignancy. The section, which is open to

inspection, shows numerous transverse and obliquely cut papillomata, and in the stroma surrounding them are thickly packed masses of nævoid, epitheliomatous (or as has been suggested, "endotheliomatous") cells, containing melanin.<sup>1</sup>

#### DISCUSSION.

Dr. J. H. SEQUEIRA (discussing Dr. Semon's case) said he had a case of such a lesion on the hand, in one of his X-ray workers at the hospital, ten years ago. The growth was removed, and the man still remained well. If in this case it was intended to remove a piece for a section, it would be as well to remove the whole growth.

Dr. L. SAVATARD said that these patches of melanoderma frequently became malignant, and that they should be treated as liable to become so. He had had a case with the lesion in the same position as in this case, and he froze it. Apparently it cleared up. But the patient returned eighteen months afterwards with raised ulcerated and slightly pigmented recurrent growth. He excised it, and the pathologist's report was that it was sarcoma. It healed up quite satisfactorily, and the patient did well. He had excised many pigmented moles, and tried, at different stages, but without success, to trace the start of malignancy. Dr. Graham Little's section shown to-day he did not consider malignant; it presented a pretty picture of a small pigmented nævus.

#### Case of Sclerodermia.

By M. G. HANNAY, M.D.

THIS patient, a man aged 68, is suffering from a definite but mild degree of sclerodermia of the diffuse type involving the skin of the whole body with the exception of the extremities. The stiffness of the skin was first noticed in the neck about two years ago, and gradually extended. During the last two months there has been a definite improvement. This may be partly due to the administration of thyroid in small doses, and partly (latterly) to rest in hospital.

Through the courtesy of Dr. Izod Bennett, the patient was admitted to the Middlesex Hospital for the investigation of his basal metabolism, which was kindly undertaken by Dr. E. C. Dodds. While in hospital the temperature always remained subnormal; pulse ranged from 60 to 80. His basal metabolism was found to be 25 per cent, below normal. Excretion of sulphur during twenty-four hours showed no divergence from normal.

In addition to the sclerodermia, there are very numerous small tags of skin, some vascular nævi, and seborrhœic and pigmented warts. Although these symptoms do not constitute von Recklinghausen's disease, they may perhaps be suggestive of a partially developed case. It is, however, from the point of view of the sclerodermia that I am showing this patient.

Dr. F. PARKES WEBER agreed that the case was one of generalized sclerodermia, but there did not seem sufficient ground for also diagnosing von Recklinghausen's disease.

<sup>1</sup> The growth was destroyed by diathermy under nitrous oxide anæsthesia in the following week.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case of Keloid after Burns.

By E. G. GRAHAM LITTLE, M.D.

THE patient, a boy, aged 11, received a burn eight months ago, and a keloidal growth has taken place on the site of the burn, and now covers almost all the middle surface of the thigh. An interesting feature is, that part of the large patch has undergone spontaneous healing. The history is very precise, namely, that the fenestrated areas which are now, apparently, unaffected, have been keloidal, and the upper margin of the keloid was marked by this little isolated lesion on each side of the thigh. At the same time, parts of the tumour formation are extending in the typical crab-like way, so this presents a remarkable combination of the retrocessive and the advancing types of the affection. I propose to have the main area treated with radium, because it is very itchy and painful, and the boy's rest is badly disturbed by it.

### ? Idiopathic Hæmorrhagic Sarcoma of Kaposi.

By HENRY MACCORMAC, C.B.E., M.D.

THE patient, a man aged 59, developed the condition seen on the feet two years ago. Some years previously he had had phlebitis, but an interval elapsed during which the lower extremities remained to all outward appearances normal. The present eruption is confined to the feet. On the dorsal surface there are superficial ulcerations or abrasions; they are not extensive, and probably arise from coccal infection. All the toes of both feet are distinctly swollen and "infiltrated": the degree of congestion is considerable at times, rendering them almost plum coloured. During the summer the lesions on the toes lessen in degree, but never disappear. It is interesting to note that the patient is of Semitic origin and comes from Galicia, two points which, taken in conjunction with the morbid appearances, suggest the possibility of a hæmorrhagic sarcoma.

#### DISCUSSION.

Dr. J. M. H. MACLEOD said that this condition appeared to him to be more like a varicose ulceration, due to some obliterative condition of vessels, than a hæmorrhagic sarcoma.

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Dr. J. H. SEQUEIRA remarked that he did not see any striking resemblance to the so-called hemorrhagic sarcoma. In the cases he had seen there had always been a chronic purplish congestion of the extremities, in addition to the formation of nodules, and in one patient still under observation, who was shown at the International Congress in 1913, the purplish congestion had persisted throughout, and the granulomatous nodules had been more of the nature of an epi-phenomenon. The late Sir Jonathan Hutchinson originally described the condition as chronic symmetrical purplish congestion of the extremities. This patient's upper extremities were free. Histologically, the condition was inflammatory, and was best described as an angiomatous granuloma. Dr. MacLeod's suggestion was a more probable diagnosis.

### Two Cases for Diagnosis.

By HENRY MACCORMAC, C.B.E., M.D.

*Case I.*—This boy, aged 14, has had the present condition four months. The eruption began on the chest, and gradually spread, so that it is now extensively distributed over the thorax and abdomen, upper and lower arms and thighs, and is present to some extent on the face. There are no subjective sensations, and no lesions have been found on the mucous membrane; the lymphatic glands are not enlarged. Some of the lesions are small maculæ with scaling; some are distinctly papular.

*Case II.*—Patient, a girl, aged 18, with a similar eruption. In her case the lesions first appeared six months ago, are profuse, and distributed in an identical manner. The greater part of the eruption is of the erythematous-squamous type and probably none of the lesions is papular, although in some the suggestion of a papular nature is conveyed to the observer. The elbows and knees are not affected.

In both cases the Wassermann reaction was negative.

These two cases present a peculiar erythematous-squamous eruption, four examples of which have recently come under my observation. In the first two cases seen the lesions completely cleared up after remaining present for from four to six months, and I think in the present cases a similar course will be pursued. It may be added that the second (female) patient was admitted to my ward with a diagnosis of psoriasis. Treatment seems to have but little effect on the lesions, and judging by the previously seen cases it would appear that the eruption follows a prolonged course, in time disappearing spontaneously.

#### DISCUSSION.

Dr. J. H. STOWERS inquired why Dr. MacCormac doubted the seborrhœic origin of these cases as, in his opinion, they were of that nature.

Dr. H. G. ADAMSON (President) thought that both were cases of acute psoriasis.

Dr. HALDIN DAVIS regarded both these cases as belonging to the group of pityriasis lichenoides chronica. He could not agree with the view that they were psoriasis. He did not think that any case, even of acute psoriasis, would last so long as these had—five or six months—without an increase in size of the individual lesions or without showing any tendency of the lesions to coalesce. Moreover it was not possible to get a typical psoriasis scale off any of the lesions. He also thought that the long duration of the cases proved that they were not pityriasis rosea.

Dr. BARBER agreed with Dr. Haldin Davis in regard to the second case.

Dr. DOUGLAS HEATH expressed his agreement with Dr. Stowers' views.

Dr. GRAHAM LITTLE said he had recently seen a case like these, but with a much longer history, namely, some years. In that case his confident diagnosis was parapsoriasis, and he would have diagnosed the present two cases as such but for this experience. His own case cleared up completely with nothing but local treatment, and the ultimate diagnosis was that of psoriasis.

Dr. MACCORMAC (in reply) said he had only recently seen the boy. He made a section from one of the lesions in the case of the girl. It was not typical, but it suggested psoriasis.

### **Two Cases illustrating the Benefit of Light Baths in Tuberculous Disease of the Skin.**

By J. H. SEQUEIRA, M.D.

THESE two boys, suffering from a grave type of lupus, illustrate the advantages of the light bath. Much attention has been paid to the influence of sunlight on the general metabolism, and, according to the work which was done by Rollier, at Leysin, and in this country by Gauvain, as the patients become pigmented, so their tubercular lesions benefit. It has been found, in surgical tuberculosis, that under sunlight sinuses heal up, and there is a remarkable improvement in the general condition. I show these cases because we now have a practical method at the London Hospital for treatment by light, even during sunless weather.

I have been working on the method adopted at the Finsen Institute, at Copenhagen, namely, that of seating the patients around a strong arc lamp. The treatment was started at the London Hospital in July last, beginning with a small lamp, and using a larger one as we gained experience. We can now treat at one time eight of one sex, who wear only bathing drawers, the eyes being carefully guarded by thick veils, which our ophthalmologist, Mr. Goulden, considers a sufficient protection. A patient is first put under the influence of the lamp for half-an-hour, the dosage being gradually increased until he sits for four hours a day opposite the lamp. We are much impressed by the value of this method in lupus, and I have brought these two lads to show what can be done in cases which have been very resistant to treatment.

The younger boy is aged 15, and he has had lupus from his seventh year. He had been treated by inoculations of tuberculin, by creasote and salicylic acid plaster, by X-rays, and by Finsen light, and photographs of him taken last January show how extensive was the disease.

The elder of these boys came to me in September, 1921, and was treated by the usual methods until August, 1922, and the disease was practically as extensive at the end as at the beginning; we could not keep pace with the lupus process.

Both these boys are enormously benefited, physically, and even mentally, by the light bath; they have put on weight, and their blood count has improved, and the tubercular lesions have reacted in a remarkable way. Since August last they have had the light bath only.

The strong carbon arc lamp is fixed three feet from the ground, and the patients, screened from the draught, sit in a circle round the lamp, exposing back and front alternately. I commend this to my colleagues as a valuable adjuvant in treating chronic tuberculosis of the skin. Some of my surgical

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colleagues are so impressed with our results that we may have further lamps put in. It was proved in Copenhagen that the carbon arc gave better results than mercury vapour or tungsten lamps.

### DISCUSSION.

Dr. W. J. O'DONOVAN said that this new work, on which Dr. Sequeira and himself had been occupied since August, 1922, was still experimental. The cases they had so far worked at had been solely cases of exuberant ulcerating lupus of the face and neck. For the first exposures, half-an-hour, twice a day, was the dose employed for a week, then an hour twice daily for a week, then two hours twice daily: they had not exceeded this. At intervals all the patients are turned about. After a week there was erythema of the trunk with fine superficial scaling, in a fortnight in dark subjects there was deep pigmentation of the exposed skin; about this time the ulcerated areas began to dry rapidly, the protuberant granulations began to shrink up and an appearance of healing was perceptible. He must add, that all their cases were cases which had been tried and had failed under ordinary Finsen light treatment. It had early been found necessary to shield active lesions while under the arc exposure, and this dressing of lint affixed by ointment was sufficient to ward off pain; under the Finsen lamp the lesions were exsanguined by pressure, but under the stimulus of the arc light they became congested and painful. Some patients became very thirsty. In blonde patients with a fine skin these broke out early in large sheets of painless blisters, but this did not deter them from further exposures. All the patients had improved in height and weight; lethargic and untidy boys become bright, clean, and mischievous. In Copenhagen where he (Dr. O'Donovan) has seen this treatment installed on a large scale, all forms of tuberculosis, lupus, visceral and bone tubercle, were so treated at the Finsen Institute, the patients residing meanwhile in Government hostels. Wider experimentation with this new therapeutic aid was to be carried out but its utility in the worst forms of facial lupus was beyond question.

Dr. H. G. ADAMSON (President) said that Dr. Sequeira was to be congratulated on having started this treatment in the out-patient department of the London Hospital. Lupus cases were difficult to cure, largely because of the conditions under which the patients lived.

Dr. J. H. SEQUEIRA (in reply) said that the question of economy had been considered in this matter. An ordinary arc lamp which had been scrapped was brought into use and hung by the hospital engineer, and the cost of running it was tenpence per hour for treating six patients.

### Case of very Extensive Sclerodermia.

By E. G. GRAHAM LITTLE, M.D.

THE patient, a woman, aged about 40, has had a progressive sclerodermia for several years, which has advanced to occupy large areas of the chest and back of the trunk, the abdomen, and thighs. The disease had rapidly improved apparently as a result of the extraction of her teeth which were pronounced to be septic. This case is reminiscent of the very extensive case of sclerodermia which I showed to the Section several years ago,<sup>1</sup> that of a lady who had been under the care of Sir Thomas Barlow, and who had also rapidly improved out of all recognition as a result of the extraction of all her teeth.

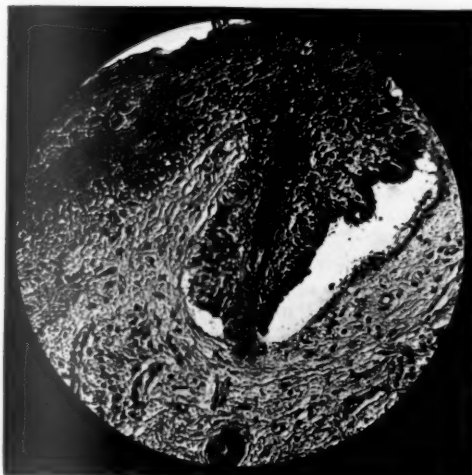
<sup>1</sup> *Proceedings*, 1916, ix (Sect. Derm.), p. 69.



**Case of *Ulcus Rodens Erythematoides*.**

By LOUIS SAVATARD.

IN October last Dr. Adamson showed at a meeting of the Section a case of "Multiple Superficial Rodent Ulcer: possible Embryonic Sweat-duct Origin," and demonstrated microscopical sections of the growth to support this theory of origin. To-day I show you sections (*see figure*) of a similar case which clearly demonstrate a sweat-duct passing through the button-like masses of



basal cells. The tumour cells take the hæmatoxylin stain more readily than do the duct cells, and under a high power can easily be differentiated. I suggest that Dr. Adamson's sections simply show the involvement of sweat-ducts in the growths.

Dr. SAVATARD also demonstrated slides and sections of the following cases :—

- (1) Peri-articular Cystic Fibromata of the Skin (the so-called "synovial" tumours of American writers).
- (2) Multiple Sebaceous Carcinomata.
- (3) Early Spino-basal-celled Carcinoma (Darier).

**Manganese as a Chemotherapeutic Agent.**

By J. E. R. McDONAGH, F.R.C.S.

(ABSTRACT.)

CLINICAL experience with arseno-benzene led me to believe that the drug did not attack the *Leucocytozoon syphilidis* directly, but indirectly through the protein particles in the plasma. Seeking an explanation of the action of chemotherapeutic drugs I came to the conclusion that metals acted as catalysts in the oxidase reaction and non-metals as catalysts in the reducase reaction. Further work led to the result that metallic preparations in general increase the number of the protein particles and augment their negative charge, while non-metallic preparations in general cause the protein particles to agglutinate and diminish their electric charge. In short, metals act as conductors of electricity and non-metals as condensers. All micro-organisms behave as condensers; their action varies, a fact which enables them to be graded. Coccogenic bacteria are not such powerful condensers as protozoa. Of the former the staphylococci are the most feeble and of the latter the *Leucocytozoon syphilidis* is the most potent. The effect of conduction is to increase the number of the protein particles, diminish their size, hasten their movement (Brownian movement) and augment their negative charge. This means that the expanse of surface exposed to the parasites is increased enormously and the additional negative charge enables the protective substance to turn the tables on the parasites. The protective substance now acts as the more powerful condenser and robs the parasites of some of their negative electricity. If the parasites lose sufficient they undergo lysis and are thus destroyed. In the test-tube deprivation of negative electricity more frequently results in the parasites undergoing agglutination and precipitation. In fact all the immunity tests are based upon the electrical relationship between the protective substance and the micro-organisms. As the conductor action of the different metals varies considerably, and, as the nature of the compound of which a metal forms part materially influences its conductor effect, it became necessary to devise simple tests to determine the degree of dispersion capable of being produced, and to try several compounds first in simple and then in complex infections. The increase in the number of the protein particles can be detected with the ultra-microscope. If the increase in number becomes sufficiently great intravascular clotting is caused. Consequently, some idea of the conductor action of a metallic preparation can be judged by its ability to accelerate coagulation. Conductors part with their electrons, and these, when taken up by Gram-positive micro-organisms, change them to Gram-negative. The better the conductor the more rapid the change. Conductors raise the suspension stability of the blood and increase the percentage of blood-sugar in the plasma. Condensers, on the other hand, diminish the number of the protein particles and cause them to increase in size and agglutinate. Condensers retard coagulation. They take away electrons from Gram-negative micro-organisms and make them Gram-positive. They reduce the suspension stability of the blood and cause a fall in the percentage of blood-sugar. The most suitable conductor is manganese and in its colloidal hydroxide form it is useful in the simplest infections. With the hope of finding a preparation which would be as beneficial in streptococcal and gonococcal infections as the

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hydroxide is in staphylococcic infections I experimented with various compounds and finally found manganese butyrate to be the most efficient. The more complex a compound becomes the greater is its initial condenser effect, but when the drug becomes adsorbed by the protein particles the contained metal is liberated and is able to exert a maximum degree of conduction from being, so to speak, on the spot. Adsorption is greatest when the compound contains amino groups, and this explains why the immediate effect of arsenobenzene in syphilis is that of retarding coagulation. The additional condensation to that already produced by the disease aggravates the lesions and this is the rationale of the Herxheimer reaction in syphilis and the negative phase after vaccines. In simple infections the degree of conduction required is not great, but owing to the very rapid way in which the micro-organisms multiply there must be no exhibition of a condenser action. A 1 per cent. solution of manganese butyrate fulfils these requirements. Of this preparation never more than three injections should be prescribed in any one course, because over-dosing with conductors either drives the protein particles into true solution or causes them to undergo condensation. In my experience, two injections suffice, and these should be injected intramuscularly in 1.0 and 1.5 c.c. doses respectively, with three or four clear days' interval between them. Manganese butyrate is especially valuable in boils, carbuncles, erysipelas, whitlows, lymphangitis and lymphadenitis. The drug is equally useful in acute gonococcal urethritis, abscess formation, epididymitis, and in gonococcal septicæmia, if prescribed in time. Manganese butyrate relieves in asthma as it changes the condensation of the protein particles, which causes the condition, into dispersion. In all septic conditions including tonsillitis, quinsy, and even rhinitis, manganese butyrate should be given a chance before an incision is made. If prescribed in time the lesion will abort, while if pus has already formed a single injection will bring it to a head. The pus should be evacuated through a small incision and dry dressings and not hot fomentations should be applied. In all operations on septic bones, joints, &c., manganese butyrate will be found to expedite healing. In inoperable cancers manganese butyrate removes the sepsis and reduces the septic lymphadenitis. The toxæmias of pregnancy are the result of the condensation undergone by the protein particles in the plasma. Therefore, theoretically, manganese butyrate should prove of the greatest value. In two cases treated to date the results were distinctly gratifying. The higher fatty acid compounds of manganese have too great an initial condenser action to be of value in the simple infections, as proved by experimentation with manganese maleate and ortho-coumarate.

#### DISCUSSION.

Dr. H. G. ADAMSON (President) confessed that he found it very difficult to follow Mr. McDonagh's arguments clearly. Many of his theories were not supported by sufficient evidence, but he (the President) was not really competent to offer any material comment and he would be glad to hear remarks from others, particularly from those who were more conversant with biochemistry. As regards the practical side he congratulated Mr. McDonagh upon having obtained in the long list of cases of different types of coccic infection the good results with manganese butyrate which his theories had led him to expect. He hoped that these good results would be confirmed by the experience of others, and that manganese butyrate would prove to be of the value in the treatment of lupus vulgaris which was predicted for it.

Dr. A. M. H. GRAY said he did not pretend to understand the elaborate experiments Mr. McDonagh had made in this research, but he would like to ask a question

or two. First, was the manganese butyrate a colloidal preparation? [Mr. McDONAGH: No, it was a 1 per cent solution.] His own experience of manganese had not been so favourable as that of Mr. McDonagh. He (Dr. Gray) found that in a case of boils which responded well to manganese one could get almost miraculous results from one or two injections, and, on Mr. McDonagh's advice, he had never given more than three injections, at two or three days' interval. But he had often found that some weeks afterwards there had been a relapse, and then, at that stage, the giving of more collosol manganese produced no benefit. That occurrence did not seem to fit in with the explanation Mr. McDonagh had given. He had understood that the colloidal manganese required some peptone to keep it protected. [Mr. McDONAGH: It had none.] He was interested to hear that, because he had assumed that one was not dealing with the effect of manganese at all, but with protein shock, which had a great effect on lesions of that type, but generally failed to bring the result off a second time. A further point was the following: he gathered that these metallic preparations had the effect of increasing the coagulability of the blood, while the non-metallic preparations diminished it. [Mr. McDONAGH: Yes.] But he was under the impression that salvarsan, and neo-salvarsan, and similar preparations, diminished the coagulability of the blood, and most of those who worked in this line had probably had a similar experience. [Mr. McDONAGH: This is due not to the action of the arsenic but to the condenser action of the amino groups.]

Dr. HALDIN DAVIS said that Mr. McDonagh had once more given the profession a remedy of considerable value in the same way as he had done when he introduced collosol manganese, which he (Dr. Haldin Davis) had himself used a good deal and with gratifying results. Indeed he had been more fortunate with it than Dr. Gray appeared to have been. He recalled in particular two cases of sycosis in the pubic region, a region which it was notoriously difficult to treat, and which he had been able to cure by injections of collosol manganese. He had given two injections of 2 c.c. at intervals of a week, and then after waiting several weeks had given a second series. After having listened to Mr. McDonagh he was quite prepared to admit that manganese butyrate was probably a very useful remedy. But he failed to follow the reader of the paper in the arguments by which he supported his theory of the action of this drug. Mr. McDonagh had employed a terminology which was entirely his own. For example he had used the terms "condensation" and "dispersion" to denote some electrical change in the blood and tissue fluids, but he had not indicated in any way how he measured the electrical changes thus adumbrated. He (Dr. Haldin Davis) would like further information on this point. The author had said that a change of an electrical nature had in one direction caused a quicker coagulation of citrated plasma, while a change in another direction caused a diminution of the blood-sugar. He (the speaker) was unable to follow the correlation between those changes and the postulated electrical phenomena. His opinion was that Mr. McDonagh very likely had found a useful remedy, for which he would receive the gratitude of the Members; but he thought that the discovery had really been made by the old method of trial and observation, and that its action was by no means due to electrical changes which had never been actually demonstrated to be caused by its administration.

Mr. H. FINKLESTONE-SAYLISS summarized the results of treatment by manganese butyrate at the London Lock Hospital as follows:—

On the first day the patient was given an intramuscular injection of manganese butyrate, 1 c.c. of a 1 per cent. aqueous solution and anterior urethral injections by potassium permanganate. Within twenty-four hours, the pain on micturition and the mental oedema disappeared, and by the third day the discharge was lessened; in some cases on the fourth day there was no discharge. On the fifth day a second injection of manganese butyrate 1·5 c.c. was given. In the majority of cases the discharge was thin and watery, in the morning only; there was no pain on micturition, and the urine was clear with a few flakes. On the eleventh day an injection of contramine 0·25 grm. dissolved in 1 c.c. of water was given intramuscularly.

Of the twelve cases treated in hospital not one had developed posterior urethritis. The average number of days on which the patients stayed in hospital until discharged, as requiring no further treatment was 51, the patient having had no discharge for fourteen days, and a urethral and prostatic examination proving negative. Palpation over metal bougie, showing no peri-urethral abscess, nor thickening of urethral wall nor enlarged follicles. Seven cases submitted to anterior urethroscopy had proved negative.

In the out-patient clinic injections were given at weekly intervals. Two injections of manganese and one of contramine were made. At the fourth week there was usually no discharge, and by the fifth week in 80 per cent. of the cases the urine was clear with no flakes. A urethral and prostatic examination was then made, which had almost always proved negative. The average attendance of fifty cases who had completed treatment, and were considered not to require any further treatment, had been 53.5 days, i.e., 7.6 attendances at the clinic. Five patients had been urethroscopied at the fifth attendance, having passed at the clinic clear urine with no flakes, and nothing pathological being found.

#### *Treatment of Acute Gonococcal Epididymitis.*

The same injections were given at similar intervals. The first injection relieved the acute pain and redness, and by the third day the testicle could be palpated without any pain being caused, also the acute hydrocele and swollen epididymis diminished in size. The temperature became normal from one to two hours later, and the patient was able to get up on the fourth day. By the eleventh day the nodule was small and painless. The local treatment consisted in the application of ung. guaiacol 10 per cent., and in the support of the scrotum by a Jullien's bandage. If the nodule was still present at the sixteenth day a second injection of contramine was given. In three cases the average stay in hospital, until no further treatment was considered necessary, was forty-nine days.

A comparison of one series of cases treated only by local and internal remedies, and of another series in which this treatment had been supplemented by detoxicated vaccines, showed that the manganese and contramine cases were free from symptoms on the average four days earlier in the resolution of the epididymis.

#### *Complications of Gonorrhœa.*

(1) *Peri-urethral and Para-urethral Abscess.*—Where definite pus formation was present an injection of manganese would hasten the pus formation and cause the abscess to point sooner. Where actual pus formation had not occurred, i.e., heat, redness, swelling, but no fluctuation, manganese tended to abort it. (2) Two cases of *para-urethral gland infection* (the so-called Tyson's gland) had cleared up in three days on injection of manganese without any incision being necessary.

#### *Sepsis and Manganese.*

Manganese had proved useful in aborting and clearing up the most resistant cases of furunculosis of the neck and thigh. It was a valuable adjunct in the control of cases of arsenical dermatitis. One case of recurrent arsenical dermatitis had developed furuncles on the thigh, also a nodular lymphangitis. One injection of manganese aborted the condition.

Mr. McDONAGH, in reply to the President, said that it was never suggested to use manganese butyrate in lupus vulgaris. He also said, in reply, that the clinical results from manganese butyrate were much superior to those obtained with manganese hydroxide. This was especially the case in the treatment of gonorrhœa. So far as the criticisms of his paper were concerned they could not be answered without his reiterating what he had just read. No words had been coined as suggested and the subject matter was extraordinarily simple, as he believed most would find it to be when reading the article at leisure. He believed he was the first to find out that arseno-benzene did not attack the syphilitic micro-organisms directly, but indirectly through the protein particles in the serum. In

order to explain this indirect action he stated that his research work had led him to believe that metals acted as conductors of electricity and non-metals as condensers. Conductors caused dispersion of the protein particles, and consequently increased the area of the protective substance exposed to the parasites. One of the characteristics of dispersion was acceleration of coagulation, but the reason why the initial effect of arseno-benzene was that of retarding coagulation was because the amino groups of the benzene nucleus acted as condensers. Condensers caused the protein particles to agglutinate, an action which resulted in a loss of negative electricity, hence the reason why the first effect of prescribing salvarsan in early syphilis was to make the lesions worse. If a powerful non-metallic substance was injected into a patient with a primary sore the addition to the condensation already produced by the disease would be sufficient to bring out the rash. Simple metallic substances were required in simple infections and complex substances in protozoal infections. But it must be borne in mind that the more complex a compound became the greater was its initial condenser action. The danger of using metallic substances was that excessive dispersion led to true solution, in which state the protein particles ceased to act as protectors. This was the reason why the recurrences of syphilitic manifestations reached such a high figure when one course of injections was deemed sufficient. Also, why so many of the recurrences were of the nature of a chancre redux. He had investigated a large number of cases and was of the opinion that none were cases of reinfection. In the case of manganese butyrate in coccogenic infections clinical experience clearly showed that not more than three injections should be prescribed in any one course. If this number were exceeded the appearance of fresh lesions would be stimulated. Recurrences definitely showed that the action of treatment upon micro-organisms was indirect.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

### Case for Diagnosis.

By A. WINKELRIED WILLIAMS, M.B.

PATIENT, a schoolboy, aged 13. No drug history, no evidence of food poisoning, no history of any suspicious sore anywhere on body for the last twelve months. He was sent back from school three weeks ago, with what was thought to be varicella. Three days later was seen by Dr. Le Riche, of Worthing, who at once noted the unusual features of the eruption. It began as a varicella-like eruption on chest and abdomen, where it is now fading and in some places is gone. It spread centrifugally, the last parts attacked being the soles of the feet and palms of the hands, and it is now just beginning to break out on the face and buccal mucosa. The vesicles capped red papules or macules; they dried up in a day or two leaving a thin scale which peeled off and left a bluish red papule or macule showing through the diascopos a brownish pigmentation. The rash is thickly studded over the whole body, and is slightly purpuric about the axilla and lower limbs. A week ago there was no palpable enlargement of the lymphatic glands. Yesterday some glands were hard and enlarged.

### DISCUSSION.

Dr. A. WHITFIELD thought it was a case of disease which had not yet received a name, but looked exactly like secondary syphilis. The present case was the fourth instance of the condition he had seen. He agreed it was a toxic condition; one of his own cases had coli bacilluria, and it would be well to ascertain whether other cases suffered in the same way.

Dr. GRAHAM LITTLE said he was at first inclined to regard it as a syphilide; the glands—epitrochlear and others—were much enlarged. But the vesicular origin of the condition was against that diagnosis. The tonsils also were septic, and he regarded the eruption as toxic. He had never before seen its counterpart.

Dr. J. H. SEQUEIRA referred to the case of a girl, aged 14, with a, presumably, toxic eruption similar to that in the present patient. He was asked to see her during the small-pox scare, as she was living in a crowded boarding-house. She had no temperature when he saw her, but, he understood, it rose afterwards. It cleared up under symptomatic treatment in a fortnight. She had some glandular enlargement.

Dr. A. CASTELLANI said he had seen a similar condition in the tropics; it was depicted in a plate in the work on "Tropical Medicine" by Dr. Chalmers and himself (see p. 2249). The cases he saw in Colombo were severe. There was a generalized eruption of large papules, which were of bright red colour. Sometimes the cases were mistaken for secondary syphilis, or, in Ceylon, for small-pox. Several glands were enlarged in the tropical cases.

Dr. SEMON suggested that the condition now shown might be an aberrant type of pityriasis rosea; the glandular enlargement would support that view; and on the arms, in places, there were the typical scaly rings.

P.S.—Since the meeting a Wassermann test has been done; it was negative. A differential blood count showed no abnormality.



### **Urticaria Pigmentosa in an Adult.**

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a girl, aged 20, gives the history that she began to have an itchy eruption in the same positions as the present rash, two years ago. She has now a scattered eruption of brownish-red macules on the forearms, buttocks and legs, which become slightly turgid on friction, but do not itch much. A section was taken at another hospital, where she has been under treatment with X-ray applications for some time, with no promising result as regards the lesions. The nature of the section cannot be ascertained.

Dr. M. G. HANNAY said he had a patient, a man, with a similar eruption; there was definite pigmentation, and an urticarial reaction to scratching. A biopsy, however, showed no increase of mast cells. The man was aged 36, and he had had the eruption about six years.

### **Psoriasis of Anomalous Type.**

By HALDIN DAVIS, M.B.

EIGHTEEN months ago this man damaged the middle toe of his right foot, and a scaly patch appeared on it. Then the nails, first those near the injured toe and afterwards those more remote from it, became affected. Later the finger nails were involved. He has also had scaly patches on his head. There are two areas in the groins which, on account of their position, have kept soft, and are not very typical in appearance.

Do the members agree with my diagnosis of psoriasis, and can they suggest any more effective treatment than small doses of X-rays? I have tried this both on hands and feet, but without much result; the fingers appeared to be better at one time, but then they relapsed to the former state. The best we are able to do is to keep the nails soft with emollient paste. Ringworm of the nails has been suggested, but no fungus could be found on repeated examination.

#### **DISCUSSION.**

Dr. S. E. DORE asked on what grounds the diagnosis of psoriasis was made in this case. He saw this patient privately some time ago, and examined the nails for ringworm, but could not find the fungus. He did not think there had been psoriasis on any other part of this patient's body, or that the appearance of the nails was that of psoriasis.

Dr. LESLIE ROBERTS said he had recently seen a case of psoriasis in which the lesions on the soles of the feet had been modified by pressure resulting from the falling of the plantar arch. The patient's weight was between 11 st. and 12 st., and he had been for the most part confined to the house for three and a half years. The increased pressure had modified the external aspect of the disease, so that the diagnosis of dermatitis had been made. This unusual feature was simply an exaggeration of the intracuticular leucocytosis, which occurred, probably, more or less, in every case of psoriasis. Dr. Davis's case was in some respects similar to his.

Dr. H. G. ADAMSON (President) said he would not hesitate to regard this as ordinary psoriasis. There was the typical pitting of the nails, and there were typical psoriasis patches on the groin and scalp.

**Case of Urticaria Pigmentosa.**

By J. M. H. MACLEOD, M.D.

IN this particular case the eruption appeared first as the usual pinkish urticarial lesions when the child was 3 months old. I have brought the case in order to ascertain the view of the Members with regard to the aetiology of the disease, which seems to me rather an urticarial than a naevoid condition, and is possibly the result of sensitiveness to some foreign protein.

Dr. GRAHAM LITTLE said he deprecated the identification of urticaria pigmentosa with naevus chiefly on the ground that pigmentation was a very essential part of the lesion of urticaria pigmentosa, and that urticaria certainly tended to disappear spontaneously, which was certainly not the experience with any form of pigmented naevi. As regards the fact of disappearance of typical urticaria pigmentosa, he had had an opportunity of establishing this disappearance in a case which he saw and recorded in 1908, and had seen again recently. The eruption, a very extensive one, had entirely gone. As regards the diagnosis of cases as urticaria pigmentosa, especially in the adult, he was sceptical of the statement that true instances of this condition occurred with an absence of mast cells in sections. He had personally never seen such a case, and had examined very many sections derived from numerous cases in both adults and children.

**Case of Urticaria Pigmentosa.**

By J. A. DRAKE, M.D.

THIS is a case of typical urticaria pigmentosa in a baby. It shows very well the turgescence of the lesions when irritated by clothing or movements; the condition is extremely irritable.

**Case of Boeck's Sarcoid.**

By J. L. BUNCH, M.D.

PATIENT, a woman, aged 35, has a large number of nodules on her face, varying in size from that of a millet seed to that of a pea. They are irregularly scattered on the cheeks, chin, forehead, and round the orbits. They are of a pinkish, or slightly yellowish tint, definitely raised above the surface, firm to the touch, but two or three show a slight tendency to pustulation and, on their being pricked, a minute drop of pus can be exuded from them. The section under the microscope shows numerous giant cells and there is small-celled infiltration. Although the disease is usually described as the sarcoid of Boeck and is said to be related to bovine tuberculosis, I regard its distinction from acne agminata and acnitis as rather problematical.

The lesions first appeared on the chin nine months ago and when I first saw the patient the nodules were larger here than elsewhere. As can be seen to-day the chin nodules have practically disappeared, only some scarring being left. This is the result of zinc ionization and I propose to treat the other nodules by the same means. I tried X-rays in repeated doses before resorting to ionization, but the nodules were quite refractory to X-rays.

## DISCUSSION.

Dr. GRAHAM LITTLE had little doubt that this was a case of acne agminata with all the marks of the group as described by Crocker, the multiplication of lesions in the sulci about the nose, the small translucent papule with little necrosis, being especially characteristic. This condition was to be distinguished from acute miliary lupus, which was a much rarer affection, associated with true bacillary tubercle.

Dr. A. M. H. GRAY said that some years ago he went into the literature of this subject, and it was very difficult to come to a conclusion as to whether the cases originally described by Barthélemy as acnitis were the same thing as Crocker's acne agminata; he thought they were not. He did not doubt, however, that the condition Crocker described was the same as the miliary sarcoid of Boeck. Looking at the description of the original acnitis cases, pustulation was a marked feature, while in acne agminata pustulation, if it occurred at all, was quite a secondary phenomenon, probably due to blocking of infected pilo-sebaceous follicles. It struck him that probably the original acnitis cases were true tuberculides; whereas the cases such as the present one belonged to a different class altogether. The most remarkable point about Boeck's sarcoids was the absence of any tendency to break down; and if one examined sections histologically, there was an extraordinary absence of endothelial proliferation. Had Dr. Bunch tried salvarsan injections for this condition? Boeck's sarcoids disappeared in a striking manner under salvarsan.

**Trichorrhexis Nodosa.**

By J. L. BUNCH, M.D.

ALL the hair of this girl has broken off at practically the same length; in fact one would have said, from the appearance, that her hair had been "bobbed." She has also nearly lost her eyebrows: i.e., the hair has broken off short. A further point is, the mother says that unless the head is thoroughly washed at least three times a week, all the head hair becomes so matted together and arranged in bundles that the school authorities will not admit her to school. Strong soda and soaps, such as "Sunlight" soap, have been used to try to get rid of the obvious pediculosis and may have had something to do with the hair becoming brittle and breaking off short, but the trichorrhexis nodosa has been present a long time—for years—and the pediculosis is, almost certainly, of recent origin, or it would have been attended to by the school authorities.

Dr. KNOWSLEY SIBLEY said he had under his care at the present time a similar case in a single woman, aged 36. She had had the disease for three years, and the majority of her hairs showed typical trichorrhexis nodosa structure under the microscope and her hair, especially over the occipital region, broke off at about half an inch long. He had tried every kind of antiseptic lotion without result. He then gave her a full pastille dose of X-rays over the occipital region, and removed all the hairs by the roots but the new hair which grew showed the same structure. At present she was being treated with ultra-violet rays, and there seemed to be some improvement. Recently she brought her sister, who was 2 years older than she and whose hairs presented the same trichorrhexis condition under the microscope. She had noticed this complaint coming on for about a year. The sisters lived in the same house but did not sleep in the same room, nor ever did they use the same brush or comb.

**Case for Diagnosis.**

By ARTHUR WHITFIELD, M.D.

THIS child was first brought to the out-patient department when a few weeks old and the condition as then seen was in all probability congenital. At that time the salient features of the case were as follows: The child was very puny and small but otherwise no evidence of ill-health was obtained. The cheeks were swollen, tense, and of a bright vermilion red with a polished surface. In the centre of the red area on the right cheek there was an oval white patch. On pressure the colour disappeared to a great extent and revealed the fact that there was a certain amount of naevoid telangiectasis underlying the diffuse redness. The hands were intensely cyanotic and slightly swollen but at that time no naevoid growth could be detected. Practically the whole of the gluteal region was in the same condition as that noted on the cheeks. The feet were not affected.

Owing to the cyanosis and general condition I have administered small doses of thyroid gland and during the period succeeding the administration a marked change has taken place. The cyanosis has disappeared and the general condition of the child has improved. The cheeks have lost the swelling and tension but as a result the naevoid condition has become more evident. The hands have ceased to be blue but now show a naevoid condition which has extended up the wrists and is apparently dying down slightly, leaving some pigmentation. The buttocks have undergone the same changes as those in the face, and the feet and ankles have become affected and their condition now resembles that of the hands and wrists.

There has been some nasal discharge at one time but a careful examination by an expert rhinologist has revealed nothing beyond slight catarrh and this disappeared so quickly that it was thought to be nothing but a common cold.

I have never seen a similar case and when I consulted the literature I wondered whether this was an anomalous case of "erythredema" or the "pink disease." On the whole I think not and am more inclined to view it as a unique case, and am also inclined to think that the symptoms will eventually die away.

**Acarus from Case of Mange in the Human Being infected by a Dog.**

By ARTHUR WHITFIELD, M.D.

MEMBERS are all probably familiar with mange in the human being caught from the dog. Owing to the kindness of Professor Hobday, I have seen an unusually large number of such cases, but in all my experience, and after hours of hunting, I could not find the acarus. Six months ago a practitioner came to see me suffering from a condition which I diagnosed as mange. The doctor admitted he had a dog, but said it did not scratch very much, and as there was a history of food poisoning, I thought I was wrong. A week or two later the doctor wrote that his wife was affected, and that the dog was scratching. The lady then came to me bringing the dog with her and the acarus was demonstrated in abundance from the scales on the dog. After a long search I also found a burrow on the wrist of the lady, and I picked out the acarus from it.

The other day a lady came to me with a very definite history. She bought a dog from a breeder, and it was covered with mange, and shortly afterwards

she developed the typical disease, and her daughter developed it too. It quickly died out in the daughter. This patient had the typical mange aspect: that is, an erythematous-papular rash all over the body, and everywhere it was intensely pruritic: but there were no runs. I was just giving up the search, when at the tip of one elbow I found a follicle with a slightly brownish speck and it proved to be an acarus, which I have brought for comparison with a human acarus. I am unable to distinguish between the two except by size.

The dimensions of the human acarus were accurately measured and proved to be 0.33 mm. in length and 0.306 mm. in width. Those of the *Sarcoptes canis* were 0.288 mm. in length and 0.255 mm. in width. That is, the human acarus is larger than that of the dog roughly in the proportion of five to four, a difference which would render it difficult to distinguish between the two without measurement.

The lesion by which I made the diagnosis was this: If one could imagine a varicella lesion divided by about ten in size, it would be exactly like a mange lesion, i.e., a very fine oval erythema, in the centre of which is the smallest vesicle discernible with the naked eye, smaller than a pin head. I think that probably the acarus commonly creeps into the neck of the hair follicle. According to the statement in books, if untreated the disease dies out in six weeks.

#### DISCUSSION.

Dr. WILFRID FOX said these cases were commoner than many supposed. He had recently seen a case which he traced directly to a dog which had ordinary parasitic mange, and which had infected both husband and wife: the lesions were of the type so accurately described by Dr. Whitfield. From the point of view of treatment, he did not think it mattered which variety of acarus it was.

Professor HOBDAY congratulated Dr. Whitfield on having found the parasite in this case. Being engaged in canine practice he saw twenty or thirty cases a year in which people were definitely infected from the dog. Quite recently he had had two cases in medical men. He did not think it was as well known as it ought to be that mange in the dog was responsible for a large number of cases of irritation of the skin in human beings where dogs were kept as intimate pets. In Yorkshire a medical officer of health bought a pug dog which had mange badly, and it contaminated all the members of the family before the source of the trouble was recognized. The front of the forearm, where the skin was very thin, was a common site of infection, and dogs were often allowed to rest on the forearm when being nursed.

Dr. A. M. H. GRAY asked whether Dr. Whitfield had ever succeeded in getting the larva of the human acarus out of the papule of human scabies. He (Dr. Gray) had once managed to remove a larval acarus out of a follicle which was at some distance from the main run. He believed that the ordinary urticarial eruption of scabies was due to the larvæ getting into the hair follicle, and setting up irritation in that site.

Dr. WHITFIELD (in reply) said that he had never found the larvæ. He tried on several occasions some years ago without success, but after seeing this case he might succeed better. Only once had he picked out a male acarus. Unlike the sluggish female acarus this darted about quite quickly in a drop of water under the microscope.

### Two Cases of Angiomatous Granuloma (Multiple Idiopathic Pigment Sarcoma of Kaposi).

By J. H. SEQUEIRA, M.D.

Case I.—S. F., tailor, aged 56, whom I showed originally in 1913, an account of his case being published in that year.<sup>1</sup> The patient, of Hebrew

<sup>1</sup> *Brit. Journ. Derm.*, xxv, p. 351.

extraction, was born in Poland, coming to live in the East End of London in 1905. The illness began in 1911 with purplish swelling of the feet and when seen in 1913 the left wrist and hand were affected. There was considerable non-pitting infiltration of the skin, which was so dense that the movement of the toes was impaired. In addition to the general purplish infiltration there were discrete papules of similar colour outside the general infiltrated area. The Wassermann reaction was negative. Dr. Turnbull, in an exhaustive report on the microscopical appearances (*loc. cit.*) showed that the chief abnormality was an increase in the number of the capillaries, this increase apparently being due to an actual proliferation and not merely to a congestion rendering the capillaries more conspicuous. Round and between the capillaries there was a slight proliferation of spindle fibroblasts and a slight infiltration by mononuclear basophil cells. The majority of those cells appeared to be free endothelial cells, a few resembling lymphocytes. There were no leucocytes and no plasma-cells. In another case reported upon by Dr. Turnbull there was much intra- and extra-cellular pigment giving the iron reaction, the deposit of pigment being doubtless a secondary phenomenon due to hæmorrhages from engorged capillaries.

The patient, who still shows the purplish swelling of the extremities, has been under my care for nine years and from time to time raised flat purplish swellings have been observed on the general infiltration. He has had occasional treatments by X-rays and these have materially diminished the swelling and have, thereby, relieved the stiffness and occasional pain. The patient's general health has not suffered. He is still well-nourished, though anæmic. There has been no evidence of gout.

Case II.—D. S., tailor, aged 61, was admitted to the London Hospital on February 7, 1923. He is of Hebrew extraction and was born in Poland. At the age of 16 he came to London and has lived in the East End for forty-five years.

Two years ago the fingers of the left hand became swollen and he found difficulty in moving them in the morning. The swelling spread to the dorsum of the hand and to the palmar aspect of the fingers. The swollen areas became purplish in colour. A year ago a similar condition appeared in the right hand and on both feet. On both hands and feet there are numerous irregularly shaped raised purplish areas with sharply defined margins. On the dorsum of the hands there are isolated flat papular lesions in addition to the confluent irregular areas. The fingers are swollen and purplish in colour and the movements are impaired. On the feet the affected areas are mainly on the dorsum, especially along the margins. They are definitely infiltrated, of a dark purplish brown colour with sharply defined slightly raised edges. The toes are not affected. The general health is good, and there are no physical signs of visceral disease. The Wassermann reaction is positive but there has been no improvement under anti-syphilitic treatment. The blood-count shows no abnormality and blood cultures proved sterile. There is no history or evidence of gout.

Histologically the lesions have the same characters as those described above; there are the same increase in the capillaries and cellular infiltration with considerable deposit of pigment giving the iron reaction.

The condition is obviously not sarcoma and I have long held the opinion that Kaposi's name, "multiple idiopathic pigment sarcoma" should be dropped. As Professor Turnbull has pointed out, the affection is an angiomatous granuloma and I suggest that this would be a convenient descriptive appellation to use until the etiology of this rare affection is worked out.



### So-called Kaposi's Multiple Idiopathic Pigment Sarcoma.

By A. M. H. GRAY, C.B.E., M.D.

PATIENT, a male, aged 67. The lesions appeared suddenly in 1915; he had a few similar lesions on the arms at the same time, but they have disappeared. The affection is not so marked as in Dr. Sequeira's cases, and the case is more doubtful in type. I think it closely resembles the cases described by the late Dr. Pringle<sup>1</sup> not long ago. They differ from the ordinary simple hæmorrhagic lesions associated with chronic vascular stasis, in that the lesions are more or less persistent, very sharply defined, and, certainly microscopically, they show the same type of change which Dr. Sequeira has described. I have a section of one of these lesions, and it shows an extraordinarily sharply marked swelling immediately underneath the epidermis, and slightly stretching it, the swelling consisting of a mass of new connective tissue cells, and a large number of capillary vessels closely resembling an angioma.

Dr. J. H. SEQUEIRA agreed that Dr. Gray's case was a rather doubtful one. In all the cases in which he (Dr. Sequeira) had been able to make a firm diagnosis, the four extremities had been definitely affected and that affection all began in the intense purple congestion.

### Case of Rodent Ulcer under Treatment with Arsenic Paste.

By A. M. H. GRAY, C.B.E., M.D.

PATIENT, a woman, aged 46, had a nodule in front of her right ear seven years ago. It was first treated with X-rays until it disappeared. It recurred, and was treated at Norwich Hospital with X-rays and CO<sub>2</sub> snow for eighteen months; it was also scraped several times. Subsequently it received about a year's treatment with X-rays at Lowestoft Hospital. I first saw her in April last, and the size of the ulcer then is shown by this photograph (exhibited). I have brought her to show how one uses the arsenic paste in dealing with these extensive ulcers. The area is first scraped very thoroughly and in certain cases portions of the growing edge are excised. The paste, which was suggested to me by Sir Norman Walker, and which consists of arsenious anhydride one part, sulphide of mercury five parts and animal charcoal one part mixed with a little spirit immediately before application, is then applied in a thin layer over the scraped area. In this case as the area was so extensive it was treated in two parts, the anterior half being dealt with first. There have been two local recurrences calling for further applications of the paste, but the whole area now appears to be quiescent. There is a large area of bare bone, practically the whole temporal fossa is exposed, and the zygoma has already separated. The rest of the bone will separate in due course, but it often takes many months.

Dr. O'DONOVAN said that treatment of rodent ulcer by arsenic paste had largely gone out of use, and it was interesting to see a form of treatment in which the old generation of surgeons had had confidence. The only alternative treatment for advanced cases of this type was an extensive surgical removal, and a successful result even then could not be predicated.

<sup>1</sup> *Proceedings*, 1918, xi (Sect. Derm.), p. 107; 1919, xii (Sect. Derm.), p. 48.



**Erythema of Face.**

By A. M. H. GRAY, C.B.E., M.D.

THIS patient, a female, aged 27, developed secondary syphilis, and on January 12 last I gave her a first injection, 0.45 grm. of N.A.B. intravenously. The same evening she became feverish, and in twenty-four hours had developed a curious rash on her face. It has now practically gone. The rash consisted of a butterfly patch on the face, a patch in the centre of the chin, patches on the sides of the neck, and a V-shaped patch in the suprasternal region. The patches stood out sharply from the skin, and were very oedematous. The temperature for several days was 104° F., and gradually subsided. She has had fairly severe albuminuria since the injection, 6 per cent. by Esbach's method, and it has not yet entirely gone. She is said to have had acute erysipelas of the face three months before, which kept her in bed for two or three weeks, but she had no fever.

I think that both these attacks are acute lupus erythematosus, and that the disturbance caused by the injection of N.A.B. was responsible for bringing out the second attack. I have never seen any description of such a condition following a salvarsan injection.

**Case of Recurrent Cellulitis.**

By H. G. ADAMSON, M.D. (President).

PATIENT, a young woman, aged 23, has had recurrent attacks of cellulitis of the face during the past two years. The cellulitis involves both lips and adjacent parts of the cheek, the gums and anterior part of the hard palate. At



each attack there is swelling of these parts with redness and tension of the skin, the redness and swelling showing a sharp margin as in an attack of erysipelas. At first these attacks occurred every two or three weeks, but the intervals have increased, and now they occur only every few months. The swelling has never completely subsided between the attacks, so that there remains a gradually increasing thickening of the lips and cheeks until the patient now presents a "leonine" aspect (*see figure*). The patient has been

examined from time to time in the throat and nose department and in the dental department, but nothing has been discovered which might be suspected as the source of origin of the complaint. There are no nasal fissures and no fissure in the lip.

Such cases are not uncommon, and I bring this case as an example, with a view to discussing the point of origin and the treatment of these cases. In my own experience no treatment has been of any avail in this complaint, except perhaps repeated doses of X-rays, which have seemed to me to diminish the frequency of the attacks. I have tried streptococcal vaccines, intravenous injections of collosol copper and intramuscular injections of collosol manganese without any good result. Although one suspects these cases to be of the nature of recurrent erysipelas, Dr. Mervyn Gordon, who has examined many cases for me by deep puncture, has in no case been able to obtain a culture of streptococcus, so that it has not been possible to give an autogenous vaccine.

Dr. H. G. ADAMSON showed several Cases of Lupus Vulgaris treated by liquid acid nitrate of mercury.

Dr. O'DONOVAN said he had made an extensive trial of acid nitrate of mercury in severe cases of lupus of the skin of the nose, cases wholly unsuitable for Finsen light; his results had been most gratifying. The scars of Dr. Adamson's cases were beautifully smooth and supple, and in an extensive scar below the right eye the absence of ectropion was remarkable.

Dr. H. W. BARBER showed a Case of ? Premycosis Erythrodermia.

Dr. J. L. BUNCH showed a Case of Adenoma Sebaceum in a Girl, aged 10.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case of Xantho-erythrodermia Perstans.

By J. L. BUNCH, M.D.

THE patient, a male aged 47, first began to develop some small irregularly shaped flattish lesions on the chest one year ago. They were somewhat irritable and slightly scaly. During the past twelve months these first patches have increased in size and many more have made their appearance. The shape of many is somewhat oval with the long axis parallel to the direction of the ribs: others are more or less circular in outline and some are quite irregular in shape. The smallest patches are about the size of a sixpence, the largest about the size of the palm of the hand. They are distributed chiefly on the chest, some on the back and shoulders.

The itchiness and scaliness led me to suspect that it was a case of premycosis, but the induration is hardly as great as would be expected in a case in which this disease has been present twelve months.

### Case of Folliculitis Ulerythematosa Reticulata.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a boy aged 5. At present he has little more to show than the superficial pitting, characteristic of terminal stages of this disease, over the malar bones and forehead. The child was seen, when he was 19 months old, by Dr. MacKenna, who has kindly sent me the notes he then made. His diagnosis was: "An unusual variety of follicular hyperkeratosis with slight perifollicular erythema." Several of the follicular orifices showed spiny central projections but no suppuration. There were no subjective symptoms.

There have been very few cases of the disease. I think I showed my first case here, and I do not know of another instance having been recorded. Eighteen months ago Dr. Darier had a case in Paris, and he sent me a description of it. McKee, in his original paper, reported three cases. As there are very few symptoms, probably a number of cases have been overlooked. All the cases have been in young children, a slight majority of them in males. There was a history of tubercle in a small majority of the cases.

## DISCUSSION.

Dr. G. PERNET said he had recorded a similar case which he called atrophoderma reticulata symmetrica faciei. At that time he did not know that the condition had been previously described. The patient was a girl aged about 8.<sup>1</sup>

Dr. KNOWSLEY SIBLEY said he had seen two cases of this condition at St. John's Hospital. One was in a girl, aged 18, who came to him two years ago with a very marked reticulate condition over her cheeks. At that time he could not properly name it, and he called it dermatitis reticulosa. He thought it was congenital, as the mother said the child had had red cheeks all her life. Sutton's book showed a photograph which was typical of his (Dr. Sibley's) case. The patient was treated by X-rays—some eight one-third pastille doses extending over about nine months—a few months later he sent for her to be photographed, but the condition had so much abated that the photographer said there was nothing left which would show in a photograph. The other case was in a young man, aged 18, who had the same condition of the cheeks, but not so marked as in the girl. His case was complicated by a good deal of acne in the lower part of the face, the remains of the old reticulated trouble being over the malar processes.

Dr. H. C. SAMUEL asked whether telangiectasis was a feature of these cases of McKee's disease. Some years ago he showed a case of what was generally agreed to be McKee's condition, and that woman had marked telangiectases. The diagnosis lay between lupus erythematosus and McKee's condition.

Dr. H. W. BARBER said that Sir Cooper Perry had shown cases of the condition many years ago, and he (Dr. Barber) did not think the disease was so rare as Dr. Little seemed to consider it.

Dr. LITTLE replied that he did not think telangiectasis was a feature of the cases which had not been treated with X-rays.

Case of *Mycosis Fungoides*.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a male aged 50. The condition, for which he saw me a month ago, is said to have begun with a mosquito-bite on the chest. It is probably a very typical case but the diagnosis is so important that I wish to have the support of the Section. It is furiously—agonizingly—itchy. There is much infiltration in the older patches, not so much in the newer ones. It has never receded, and has always extended, so that at the present time almost the whole trunk and limbs are occupied by large patches of infiltration. No tumours have yet made their appearance, but there is general glandular enlargement, especially in the groins.

## Abnormal Scarring after Chicken-pox.

By R. TRAVERS SMITH, M.D.

PATIENT, a boy aged 14, came to me to be examined three weeks ago. He has scars, some the size of a threepenny-piece, some larger, on the abdomen and back. When 16 months old he had chicken-pox; the lesions took a long time to heal, and the doctor is said to have put on them something

<sup>1</sup> *Med. Press and Circ.*, May 31, 1916, p. 487.

which looked like paper; when this paper was taken away an ulceration was noticed, and that ulceration left scars. The boy has never been vaccinated.

#### DISCUSSION.

Dr. J. H. STOWERS said that possibly the scars, which were circular, of unequal size and distinctly cribriform in character, might be the result of vaccinia contracted in an unsuspected manner at the age of 16 months, when the patient was said to have contracted the eruption from a recently vaccinated infant.

Dr. WHITFIELD suggested that this boy had had varicella gangrenosa infantum. It might be a secondary infection on varicella, or it might have nothing to do with varicella; possibly it was a form of ecthyma. In a number of cases the condition supervened on varicella, and when that occurred the ovoid form was lost: it became a large discoid lesion, almost at once.

Dr. DORE agreed with Dr. Whitfield that the scars were probably due to gangrenous chicken-pox.

### Two Cases of Lupus Vulgaris with Treatment.

By HENRY MACCORMAC, C.B.E., M.D.

SOME time ago in a number of cases of lupus attending the Middlesex Hospital the patients were given potassium iodide in large doses. This was done on the theory that the tubercle bacillus might be an acid-fast streptothrix, a view held by certain observers. The results were not uniform, and in the majority of cases little or no benefit took place. But in one of the earlier patients subjected to this treatment very marked improvement occurred. She is shown to-day as the first case. Investigations were made by Dr. Kingsbury into the complement fixation of the series of cases, three antigens being used, actinomyces bovis, human tubercle bacillus, and an acid-fast streptothrix. It was found that in one case—that of the first patient shown to-day—a partial fixation to the acid-fast streptothrix occurred. The second case shown has also made considerable improvement under potassium iodide, and it was found that in this case there was also a partial positive complement fixation to a streptothrix. The histories of the two cases are as follows:—

*Case I.*—Female, aged 27; lupus developed on the face soon after birth, and on the buttocks at 14 years of age. She came under my care two years ago, and was put under large doses of iodides. She made marked improvement, but I found she had been previously under the care of the late Mr. Kellock, who had been using a similar method of treatment. She had thus been for a considerable time under the influence of iodides. There is now very little evidence of active disease, the site of the former extensive lesions being replaced by a good scar.

*Case II.*—Female, aged 16; the disease began six years ago, following suppurating glands in the neck, and spread over the face, involving the cheeks, nose, forehead and neck. There is also a patch over the left deltoid region which has apparently been cured by X-rays. This patient has been under treatment with iodides for some five months, and although there is still a good deal of disease present, a very definite improvement has taken place.

The patients in both cases have had some local treatment in addition to the

## 84 MacCormac: *Two Cases of Lupus Vulgaris; Case for Diagnosis*

iodides, but it may fairly be assumed that the improvement has in the main been due to the drug. The dose given has been 40 gr. of potassium iodide three times daily. This has been well tolerated.

These two cases are interesting because they both showed a positive complement fixation to a streptothrix. Mr. Kellock used iodides in other forms of tuberculosis with benefit, and it would seem that in suitable cases—that is, where either the form or the type of bacillus is different from that ordinarily met with as shown by the biological reaction—prolonged treatment with iodides may be attended with a considerable degree of success.

### DISCUSSION.

Dr. KNOWSLEY SIBLEY said he had treated many cases of lupus with iodide of potassium, but he did not give large doses; he gave it in the form known as nascent iodine. The patient took 20 gr. iodide of potassium dissolved in a tumblerful of water after breakfast, then successive drinks of 1 oz. of chlorine water in home-made lemonade, four, six, eight and sometimes ten hours afterwards. He (Dr. Sibley) did not believe in large doses of iodide of potassium; the more iodide of potassium that was given, the less was absorbed, and absorption was facilitated by giving small doses; 4-gr. doses he would regard as the maximum at one time. He had found this method excellent in the direct treatment of buccal mucous membranes, dusting diluted powdered iodide of potassium on to the ulcerated surface, then telling the patient to take some chlorine water into the mouth, and to hold it as long as possible, then to swallow it.

Dr. H. G. ADAMSON (President) said that iodine was a very old remedy for lupus vulgaris. It had been recommended by Liveing and, earlier, by the French physician, Lugol, after whom Lugol's solution was named. He (the President) had been giving Lugol's solution to lupus patients for several years past, and had given sometimes as much as 3 gr. of iodine three times daily. But in only a few cases had he seen any marked improvement, though in the "strumous" type of tuberculous skin affections and in tuberculous glands the improvement was often striking. In lupus vulgaris he had never seen any improvement so pronounced as in Dr. MacCormac's cases.

### Case for Diagnosis.

By HENRY MACCORMAC, C.B.E., M.D.

PATIENT, a male, aged 40, gardener by occupation. His duties include the care of a pony, which, he states, suffers from some form of skin disease. The condition of which he complains began about fourteen months ago with a "scab" on the back in the region of the lumbar vertebræ. The lesion resisted all forms of treatment, and finally some form of operative procedure was carried out. In this region there is now a central crust covering a superficial ulcerated area about the size of a shilling, and surrounding it there is a scar some 3 in. in diameter. Beyond this again there is an area of pigmentation and telangiectasis. No X-rays have been used.

About one month after the appearance of the lesion on the back, the arms, head and legs were affected. On the arms and legs there are now well-marked areas, some small, some extensive, sharply circumscribed with some atrophy in the more extensive lesions. At the periphery vesicles can be detected. In addition, on the legs there are a number of superficial erosions or ulcers presenting the appearance seen in a coccid infection. The Wassermann reaction is negative, and no ringworm fungus has been detected.

A small portion from the edge of one of the lesions was removed for microscopical examination. The section showed collections of cells about the vessels of the corium and also well-marked superficial vesiculation.

### Case of Recurring Erysipelas.

By J. E. R. McDONAGH, F.R.C.S.

PATIENT had a primary sore on skin of penis in 1915. Treatment with three injections of salvarsan and six of mercury was instituted before the symptoms of the generalization stage appeared. The treatment was not continued.

September 22, 1920: Patient appeared with a recurrent primary sore at the left peno-scrotal junction, and a generalized syphilitic eruption. The usual two years' treatment was prescribed and carried out.

October 10, 1920: Patient had a rigor and fever, and developed erysipelas of the penis and scrotum.

The second attack was in June, 1922, the third in September, 1922, the fourth in December, 1922, and the fifth in 1923. The second and other attacks were not ushered in with fever. Each attack has left the penis and scrotum more elephantiasic than before.

This is not a case of syphilitic lymphangitis with fibrosis, but a case of recurring erysipelas due to the streptococcus of Fehleissen. The point of entrance of the streptococcus was on the site of the second sore. It can be recognized by a multiple vesicular lesion not unlike the commencing stage of herpes febrilis. The streptococcus has been found, and has recently been cultivated after considerable difficulty and a vaccine is now being prepared, as all other methods of treatment have proved unavailing.

### DISCUSSION.

Dr. H. G. ADAMSON (President) thought that the condition called elephantiasis and leontiasis was generally of streptococcal origin, and due to repeated attacks of cellulitis (recurrent cellulitis), when it occurred in association with syphilis or with tubercle, and always when it occurred independently of these two diseases; but the theory that elephantiasis associated with a syphilitic lesion was invariably of streptococcal origin, was not in accord with the fact that in syphilitic cases the swelling sometimes subsided under antisyphilitic treatment.

Mr. McDONAGH replied that he had brought the present case in order to show that many of the so-called cases of syphilitic lymphangitis were really of streptococcal origin. The differentiation was simple, because in streptococcal lymphangitis although the streptococcus gained entrance through a destructive syphilitic lesion, the patient had many attacks, and each attack might be ushered in by fever and by a rigor. In true syphilitic lymphangitis the patient might either have no cutaneous lesion or else one which caused no break in the surface of the skin. Syphilitic lymphangitis was a progressive condition, and one which readily responded to treatment. Streptococcal lymphangitis was not influenced by antisyphilitic treatment, and not infrequently injections of arsenobenzene precipitated an attack of erysipelas.



**Case for Diagnosis; ? Diphtheria of the Skin.**

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a lady, aged about 65, for some four years past has had almost persistent crops of pustules and vesicles always covering the same parts, i.e., both feet and the left hand, including all the fingers and the nail area. The whole affected foot area is of a darkish purple colour, much infiltrated, and the skin on the soles and dorsum of the foot is considerably thickened. On the mucous membrane of the palate she has a number of bright red vesicles, and she said she had had similar vesicular lesions on the tongue and sides of the buccal mucosa. Nothing has been found in the pustules except the common staphylococcus. I only saw her this afternoon, and I brought her here. Examination of an unbroken vesicle for tinea is negative of that diagnosis.

The only similar case I have seen was one I showed here four years ago,<sup>1</sup> that of an officer, who, under the idea that his condition was syphilitic, had spent two and a half years under treatment. His Wassermann reaction was persistently negative. I had an examination made of the surface in that case, and a diphtheritic organism was found; probably the true Klebs-Loeffler bacillus. He then confirmed this suggestion by saying that at least three diphtheritic attacks had followed in his wake; he had stayed at country houses, and diphtheria had broken out after his arrival. He improved greatly with diphtheria vaccine. I think the present case may be of the same nature.

DISCUSSION.

Dr. J. M. H. MACLEOD said he thought the affection might be ringworm, because of the scaly edge of the lesion on the foot, the condition of the nails of the hand, and the fact that only one hand was affected.

Dr. H. C. SEMON agreed with the diagnosis of eczematoid ringworm. He said that recently he had had two or three such cases, but he had not been able to find the fungus. In eczematoid ringworm which became pustular, the fungus was rapidly destroyed.

**Case of Lichen Planus Atrophicus.**

By E. G. GRAHAM LITTLE, M.D.

PATIENT, a female, aged 45. There are large areas of atrophic dermatitis, covering the nape of the neck, the armpits, the area underneath the popliteal space and the side of the legs. The lesions on the lower limb are definitely raised. So although there is atrophy, it is atrophy *en plateau*—the raised area—it is of an ivory-white colour. Formerly this plateau was red. Scleroderma is an alternative diagnosis, but the history of the case and the distribution suggest to me the diagnosis I have offered.

<sup>1</sup> *Proceedings*, 1920, xiii (Sect. Derm.), p. 78.

**Case of Muriatic Acid Erosion of Fingers.**

By W. J. O'DONOVAN, M.D.

PATIENT, a man, aged 54. For twenty-two years he has worked as a galvanizer. His occupation consisted of dipping sheet-iron tanks into muriatic acid as a cleansing bath preparatory to a zinc galvanizing process. His fingers are browned and blackened by acid. There are pits and fissures in the horny palms and the finger tips are swollen, sore and corroded into a filiform appearance. For two years although still at work he has been unable to cut up his own food owing to pain and tenderness of his hands; his wife has fed him. Other men at the factory are similarly affected.

**Case of Carcinoma Faciei apud Puellam.**

By W. J. O'DONOVAN, M.D.

I AM showing a second case<sup>1</sup> of multiple early squamous-celled carcinoma of the skin of the face in a young woman. This patient is aged 21. This case is of over nine years' duration; there is a marked tendency to natural cure by scarring. The lesions as in the previous case are unilateral and below the ear running like a livid band with a well-defined raised edge along the contour of the lower jaw. A microscope section of the case is also demonstrated.

(This case will be described and illustrated in the *British Journal of Dermatology*.)

**DISCUSSION.**

Dr. A. M. H. GRAY said that these cases were very interesting, and he did not know of any description of them elsewhere. They were different, clinically and pathologically, from the so-called Pagetoid basal-celled carcinomata. Having once seen a case, the condition was diagnosable clinically.

Dr. H. G. ADAMSON (President) agreed with Dr. Gray. These growths only resembled the Pagetoid condition in their consisting of large flat patches. Dr. O'Donovan was to be congratulated on having discovered a new type of epithelioma of the skin.

**Male and Female Acarus extracted from One Burrow.**

By A. M. H. GRAY, C.B.E., M.D.

THIS specimen was obtained from the fourth toe of the left foot of a man who had had scabies for four years. The burrow was about  $\frac{1}{4}$  in. long, and with a lens two acari were visible in it. The first, at the end of the burrow, was a female, that behind it, a male. I have never before succeeded in finding a male acarus in the skin. It is generally thought that the male acarus wanders about the surface and does not burrow; but J. W. Munro<sup>2</sup> describes this phenomenon. The male acarus only comes in contact with the

<sup>1</sup> For the first case, see *Proceedings*, 1923, xvi (Sect. Derm.), p. 52.  
*Journ. Roy. Army Med. Corps*, 1919, xxxiii, p. 22.

immature female before she starts making her long burrow. The immature female and mature male burrow very closely together, or even in the same short burrow; the male impregnates the female, then the female leaves that burrow and starts one on her own account, the male never coming near her again. When she has laid all her eggs she dies. It is possible that in my case the male may have been in a tiny burrow adjacent.

### Case of *Cutis Verticis Gyrata*.

By M. G. HANNAY, M.D.

PATIENT, a male Jew, of dark complexion, age 23, has suffered since childhood from a chronic dry pruriginous affection of the skin of the trunk and limbs, but otherwise his general condition is good, and there is nothing of importance in the family or personal history. The scalp condition was only noticed about nine months ago. Over the vertex and occipital region there are



FIG. 1.

thickened folds, separated by narrow furrows, &c. (fig. 1). On the prominent portions there is a definite thinning of the hair, but otherwise the surface appears normal. There are no subjective symptoms. The condition has been sufficiently frequently described to be familiar, but no previous case has, to my knowledge, been recorded in this country. The first American cases were published last year. A section shows a mild degree of simple inflammatory reaction, chiefly localized in the neighbourhood of the follicles (fig. 2).



FIG. 2.

**Case for Diagnosis.**

By H. W. BARBER, M.B.

EVER since this patient can remember, she has had a skin which, when cut or scratched, separates, leaving, in the case of a cut, a gaping wound, and this takes longer than normally to heal; and during the last few years she has had large hæmorrhages under her skin. When first I saw her, the left arm, from the elbow downwards, was a large swollen bruise. She bleeds over her patellæ and elbows, and there are collections of blood there. Her skin feels loose like the skin of a puppy, and it can be picked up off the subjacent tissue. I thought there was a lack of subcutaneous tissue, and that the blood-vessels, being unprotected, were easily ruptured; but on excising some skin yesterday, I found there was plenty of subcutaneous tissue. She does not bleed unduly when teeth are extracted. The question of dermatitis artefacta can be excluded.

**DISCUSSION.**

Dr. DORE referred to a case of Sir Malcolm Morris, that of an elastic-skinned boy, who had congenital dislocations and who bled very easily; he had hæmorrhages of skin and joints, and small neurofibromatosis tumours. When he cut himself the edges separated and muscular tissue came into the wound. These tumours consisted of a fibrous capsule containing colloid material.

Dr. F. PARKES WEBER thought there were two kinds of so-called "loose skin" or "chalastoderma." In one there was an excess, or relative excess, of elastic elements, and the ordinary white connective tissue elements were diminished, or defective in some way, and the skin was imperfectly attached by the subcutaneous tissue to the deeper structures; this was the true "elastic skin." In the other there was a degeneration or insufficiency of the elastic elements, and an increase in the ordinary white connective tissue elements; so that, as a result of the connective tissue hyperplasia and the diminution of true elasticity, the skin and subcutaneous tissue tended to become "baggy" or to fall in a pouch-like or flounce-like manner over the parts of the body below it.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case of Disseminated Lupus Erythematosus associated with Raynaud Symptoms and Early Sclerodactylia.

By GEORGE PERNET, M.D.

PATIENT, a single woman, aged 45. I saw her for the first time yesterday, but I hope to take her into the West London Hospital for further observation. Ever since childhood she has had chilblains. At times some of the fingers go dead and then become bluish. Both little and ring fingers are permanently flexed, are more or less cyanosed, and to some extent atrophied, especially the terminal phalanges. Several of the fingers at their ends exhibit more or less circular shallow sulci (ainhum-like). The flexion of the fingers has nothing to do in my opinion with rheumatoid arthritis, but is part and parcel of the early sclerodactylia. The lupus erythematosus lesions are scattered about the fingers and backs of hands and to a much less extent about the palms. Many of them are atrophied. On the outer sides of the extensors of the forearms are a number of similar lesions. In the middle of the back there are a number of irregularly contoured islands (somewhat like the Malay Archipelago islands in shape). They consist of atrophic areas with reddened periphery. There is one small elongated oval lesion of lupus erythematosus on the back of the tip of each shoulder (quite symmetrical). About and on the ears there are reddened areas, and a palm-sized characteristic area of lupus erythematosus on the anterior part of the vertex of the head. There does not appear to be any history of tuberculosis (phthisis, &c.) in the family, but twenty-five years ago tuberculous glands were removed from the patient's neck, where there are two linear submaxillary scars, one on either side. I find nothing in the mouth. I have not had an opportunity of examining the urine. I shall have the hands radiographed in order to judge of the condition of the bones of the fingers. I have examined her left foot and found the big toe quite blue.

### Parapsoriasis—Type Xantho-erythrodermia Perstans.

By WILFRID FOX, M.D.

PATIENT, a male, aged 54, was under the late Dr. Crocker in 1903, who diagnosed his condition as stated in the above title. Dr. Crocker was anxious to show him before the old Dermatological Society of London, as a typical case, but the patient was unable to come. The condition has never cleared up during twenty years; the patient has been sometimes better and at others

worse. When I first saw him on February 21 there were numerous oval patches, reddish brown in colour, lying obliquely on the trunk in the axis of the ribs similar to the arrangement often seen in pityriasis rosea. On the arms the lesions were eczematoid and in places lichenified.

He has improved considerably since February, when I last saw him. The treatment he has received has been parathyroid,  $\frac{1}{30}$  gr., and  $\beta$ -naphthol ointment, 2 per cent.

### Case of Pityriasis Lichenoides Chronica.

By HALDIN DAVIS, M.B., F.R.C.S.

THIS is the third time that this boy (now aged 12) has been shown to the Section. He has suffered from this eruption, which has maintained almost the same state, since he was 3 months old. Dr. Graham Little showed him when he was very young, and I showed him three years ago. My diagnosis (which is also that of Dr. Little) is pityriasis lichenoides chronica, a variety of parapsoriasis, in which the lesions are very minute. The chief reason why I am showing the patient to-day is that about three months ago Dr. MacCormac exhibited a case with lesions that seemed to me exactly similar to those visible in this case, but which he thought would clear up. It will be noted by members that the lesions in my patient are minute, scaly papules, scattered over the trunk and limbs, but absent from the face, hands and feet. Many ointments have been tried, but as yet the lesions have been resistant to every treatment employed.

### Case of Congenital Onychogryphosis.

By J. H. SEQUEIRA, M.D.

I HAVE brought this child, aged 2, in order to ask assistance as to treatment of the nails. The child was born at the seventh month, and the nails were not developed at birth. When they did develop, they grew in this hypertrophic form, which resembled onychogryphosis. All the nails of both hands and feet were affected. The hypertrophied nails were strong, brownish in colour, and projecting upward from the nail beds for from  $\frac{1}{4}$  to  $\frac{1}{3}$  in. There are two other children in the family, and they are normal and healthy, and there is no history of a similar condition occurring in the child's relatives or forebears.

#### DISCUSSION.

Dr. GRAHAM LITTLE said he had had an exactly similar case; he showed it at the meeting of the Dermatological Association in London eighteen months ago. That child was a little younger than Dr. Sequeira's patient, and the nail condition was not noticed at birth. Atrophic nails developed on all the digits in the same way; previously there had been chronic impetigo. He did not succeed in getting any help in regard to treatment of that case. The nails were cut with clippers from time to time, in order to remove the local discomfort.

The PRESIDENT recommended evulsion of the nails, to be followed by scraping of the roots and nail-beds. He thought it would be necessary to destroy the nail growth entirely; the child would be less uncomfortable with no nails than with nails such as these.



## Case of Xanthoma (? Diabeticorum).

By L. SAVATARD.

PATIENT, a married woman, aged 33, first consulted me on April 10 this year. She presents numerous typical xanthomatous tumours of the skin varying in size from that of a pin's head to that of a large pea. The tumours are yellow in colour, and are for the most part discrete. They are situated over the posterior aspect of both elbow-joints, in the left anterior axillary region and on the buttocks. There is also some slight infiltration of the flexures of both palms. The patient also complains of pain in the right heel at the site of attachment of the tendo Achillis and suffers, too, from pruritus vulvæ. Occasionally shooting pains ("like red-hot needles") are experienced in the lesions, which are always painful on pressure. The tumours were first noticed three years ago in the neighbourhood of the right shoulder, and then shortly afterwards appeared in the other areas. Some lesions have been removed by the actual cautery, and have not recurred; none have involuted spontaneously. The urine has been repeatedly examined, and has always been free from sugar. At the last examination (April 13) it was acid, specific gravity 1018, and contained no albumin and no sugar. Her sugar-tolerance curve is practically normal. Dr. A. Howarth (Pathological Department, Manchester University), conducted this test for me and reports: "I have seen this type of curve in an apparently normal person at a time when he was not feeling up to the mark. . . . This is certainly not a diabetic curve." I had thought that perhaps the curve would show that the patient was a *potential* diabetic, but it does not suggest even that.

As a child the patient suffered from recurrent attacks of "gastric ulcer." In 1914 (at the age of 24 and two years after marriage) she was operated upon for a perforated appendix, and at the same time a hysterectomy was performed and her appendages were removed. In 1921 the patient saw a consulting surgeon, who diagnosed a twisted kidney. The family history is bad; her mother died, aged 48, from diabetes; her father, aged 67, from heart disease; two brothers, aged 39 and 29, from phthisis; a sister died from abdominal tuberculosis; and another sister, aged 47, from phthisis. There are two sisters alive and well.

Although, therefore, this case presents no evidence of diabetes, I anticipate, in view of her family history and of the records of other similar cases, that diabetes will supervene later, and therefore suggest that this should be classed as a case of xanthoma diabeticorum.

I should like to have the opinion of members as to the treatment of this condition.

## DISCUSSION.

Dr. F. PARKES WEBER said he did not think that in cases of nodular xanthoma of this kind anyone was justified in saying that probably diabetes mellitus would supervene, especially if ordinary care in diet were taken; there were not sufficient known facts to justify such a view. But he quite agreed that the xanthoma in Dr. Savatard's patient belonged to the same class as that which occurred in glycosuric and diabetic patients.

Dr. A. WHITFIELD said it was probable that in future xanthoma diabeticorum and xanthoma tuberosum would be merged into one classification. It was, apparently, clear that most cases of xanthoma tuberosum had cholesterolaemia, and in cases of

diabetes which developed xanthoma diabeticorum there was also cholesterolaemia. And it was probably associated with that side of the function of the pancreas, rather than with the sugar part of that organ. In the case of Harrison and himself, Harrison found evidence that the proteolytic side of the pancreas was working badly; and the speaker thought it was so in the present case too.

Dr. GRAHAM LITTLE said he had seen a very interesting clinical case, almost exactly like this, of a surgeon who had similar xanthoma lesions on elbows and buttocks. They were treated with X-rays, and for a time they improved. When he was seen, there was no glycosuria at all, but eventually he died with acute diabetic coma.

Dr. A. M. H. GRAY expressed his agreement with Dr. Parkes Weber and Dr. Whitfield. And he did not think there was evidence yet that cases of cholesterolaemia seen were necessarily associated with hyperglycaemia, or with pancreatic disease. He supposed there was no doubt the lesions were due not to hyperglycaemia, but to cholesterolaemia.

Dr. SAVATARD also exhibited Photographs and Sections of a Case of Multiple Epidermoid Cysts.

### Case of Urticaria Pigmentosa.

By H. W. BARBER, M.B.

PATIENT, a male, age 25, first noticed the eruption on his chest in 1918. Six months ago it spread to the back and arms, after an attack of severe indigestion, probably caused by eating tinned food while on a river holiday. He now has a profuse eruption of urticaria pigmentosa all over him, and the wheals are visible at places where he has scratched and rubbed. There has recently been published a case, with discussion, shown by Dr. Simon in Paris, in which the eruption appeared for the first time when the patient was 52 years of age. Two interesting facts were brought out in connexion with it. One was, that the eruption appeared after the patient had had severe indigestion, with fever—and it was probably typhoid fever, because later he developed a typhoid abscess in bone. The other point was, that he was syphilitic.

I think it is questionable whether adult cases are of the same nature as those in children. I think the ordinary infantile urticaria pigmentosa is a naevoid condition, and I believe Darier shares that view. I think the adult form must be a toxic eruption, and if one were to collect cases and investigate them systematically, the cause might be discovered.

Dr. MACLEOD said that, in the absence of a definite finding of mast cells in the tissue, it did not seem to him that a diagnosis of urticaria pigmentosa was warranted.

*Postscript.*—Histological examination of an excised lesion showed the presence of large numbers of mast cells.

### Case of Unusual Localization of Ichthyosis.

By H. C. SEMON, M.D.

THIS child, aged 5, was born with ichthyosis of the lateral aspects of the neck, flanks, and slightly on the back. The condition has persisted and leads occasionally to friction and drying effects in these areas. There is no familial

history of a similar condition in the parents or their near relatives. The interest of the case lies in the very unusual localization.

This case seems to belong to that variety of bilateral symmetrical type which finds a rare but highly characteristic expression in *keratosis palmaris et plantaris hereditaria*, or *tylosis*. Cases of this kind seem to supply rather strong evidence against the theory of thyroidal causation or influence in production. A cretin with *ichthyosis* has yet to be recorded.

Dr. F. PARKES WEBER thought Dr. Semon was right in suggesting that *hyperkeratosis palmaris et plantaris* might be allied to various kinds of congenital *hyperkeratosis* elsewhere. He thought there was a large group of congenital or developmental *hyperkeratosis*, including various forms of congenital *ichthyosis* and various forms of *hyperkeratosis* affecting the hands and feet. Sometimes the *hyperkeratosis* was of punctate distribution. He thought that in rare cases the local superficial blood-vessels were likewise involved in the congenital abnormality of growth, and that the cases described as congenital *erythro-keratosis* of the palms, &c., belonged to the same class. All these were varieties of *nævus-formation* and the congenital *hyperkeratosis* of acroteric distribution, of the "tylosis" kind, was one of the most remarkable. He thought that the case which had been shown by Dr. Haldin Davis might really be one of punctate *hyperkeratosis* of generalized distribution, and therefore might really belong to the same class as Dr. Semon's case.

### Case of Gas-burn Scarring.

By H. C. SEMON, M.D.

THIS man was very extensively burned by direct contact with the fluid of mustard gas, at Arras in 1918. The convex surfaces of the back and buttocks, from neck to thighs are involved, whilst the loins and interscapular areas appear to have entirely escaped. The scarring is regular and quite superficial, and there is no tendency to contraction, as after acid or burns by scalding or dry heat. Except for one small nodule on the apex of the right shoulder, there is no keloid formation. The scarred areas are white in colour, but pigment is excessive for several inches beyond the affected parts. The follicles appear enlarged and patulous, owing as has been suggested by the President, Dr. Adamson, to destruction of the normal, superficial openings.

### DISCUSSION.

Dr. O'DONOVAN said he had seen a man who was extensively burned in the manufacture of nitric acid, and he at first thought the case now shown was another of the same kind. The nitric acid lesion was associated with a small keloid.

Dr. H. W. BARBER said he had five mustard-gas burns on his arm and the skin was smooth. But the worst burn resembled an X-ray burn, and that took two-and-a-half months to heal, and pigmentation remained. The point of interest about mustard gas was, that, like *chrysarobin*, it was soluble in fats and not soluble in water. He found that the vapour always picked out the sebaceous areas, probably because it was fat-soluble.

**Case of Breast Tumour with Atrophy of Skin (shown at a previous Meeting).<sup>1</sup>**

By S. E. DORE, M.D.

THE following report has been received from Dr. Dudgeon:—

REPORT ON DR. DORE'S CASE OF TUMOUR OF BREAST, BY LEONARD  
S. DUDGEON, C.M.G., C.B.E.

Sections of the skin show an irregular surface which corresponds with the appearances noted on clinical examination when the skin was lying loose. There is no increase of pigmentation. A mononuclear reaction is present in the fibrous tissue adjacent to the epidermis. Between the surface layers of the corium and the deep tissue, which is highly cellular, the fibrous tissue shows a well marked hyaline change. Sweat glands and sebaceous glands are present; also numerous islets of unstriped muscle and nerve fibres. In the deepest layers large hæmorrhages are present and also well defined hæmangiomas, which could be recognized on clinical examination, and were very obvious in the deepest tissue in the portion removed for microscopy. The deepest layer of fibrous tissue is intermixed with the normal fatty tissue which it has partially replaced.

*Note by Dr. Dore.*—The general opinion, expressed by several of the members who have seen the sections, is that it is a neurofibroma.

<sup>1</sup> See *Proceedings*, 1923, xvi, p. 57.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Peculiar Folliculitis of the Scalp.

By ALDO CASTELLANI, C.M.G., M.D.

I SHOWED this patient last month, when the condition was in a much more acute stage. A number of different diagnoses were suggested: a syphilide, folliculitis decalvans, ordinary pyogenic folliculitis, lupus erythematosus, even favus. At that date I was not in a position to make a definite diagnosis, but during the intervening four weeks I have gone into the case pretty thoroughly, and will give you presently the results of my bacteriological examination. The first symptoms started eighteen months ago, and the patient was treated by a practitioner who diagnosed staphylococcus infection; he sent specimens to various laboratories, the reports from which were always to the effect that staphylococcus was present. Therefore a staphylococcus vaccine was prepared, and he has had that administered to him off and on, for the last twelve months, but without improvement. The result of my complete bacteriological examination is the following: There are a large number of staphylococci in stained preparations and on cultivation, but I succeeded in growing also a fungus, which is either a torula, a cryptococcus, or a monilia. It does not produce gas in any sugar. The cultures after a time often present a peculiar yellowish-brown pigmentation. I have little doubt that the principal part in causation of the condition is played by the fungus, and this is confirmed by the patient having been treated with a staphylococcus vaccine for twelve months without benefit. As soon as I found the fungus, I put the patient on potassium iodide and a vaccine prepared from the fungus was administered, and you see the result; the condition of the scalp is certainly much better than a month ago.

### Case of Trichomycosis Axillaris Rubra.

By ALDO CASTELLANI, C.M.G., M.D.

THIS man has been under my care for dysentery and other complaints for nearly two years. It is a typical case of trichomycosis rubra, which he appears to have contracted in this country, though he was in North Africa ten or twelve years ago. He noticed this discoloration only a year ago. Trichomycosis rubra is very rare in this country, but it is common in the Sudan, in India and Ceylon. Some years ago I separated three forms of trichomycosis axillaris—trichomycosis flava, trichomycosis rubra, and trichomycosis nigra. The first of these is the variety usually found in this country. It is caused,

according to my researches, by a minute bacillus-like fungus, *Nocardia tenuis*. The black variety is caused by the same fungus plus a black pigment producing fungus *Micrococcus nigrescens*. Trichomycosis rubra is caused by the same fungus plus a red pigment producing coccus *Micrococcus castellanii* (Chalmers and O'Farrel). Trichomycosis rubra and trichomycosis nigra are interesting examples of symbiosis; they are conditions due to two germs, a fungus and a coccus, growing together. Further particulars on the various types of trichomycosis axillaris may be found in my recent paper on the condition in the *British Journal of Dermatology*, vol. xxxiv, August-September, 1922, pp. 255-266.

### Case of Dermatitis Repens and Infectious Eczematoid Dermatitis, with Involvement of the Mucous Membranes.

By H. W. BARBER, M.B.

J. C. B., MALE, aged 58. Patient has periodically suffered from boils and carbuncles which have always quickly yielded to treatment. About three months ago he developed a whitlow on his right thumb which was opened by his doctor under gas: following the operation an eruption appeared near the base of the nail, and spread up the thumb, the horny layer being stripped up and a raw moist surface exposed. Later, other parts of the right hand became involved and the eruption also appeared on the left hand, on the penis and scrotum, on the forehead and on the feet and legs. He also developed an acute discharge from under the foreskin, conjunctivitis, and stomatitis.

He was referred to me by Dr. Flynn on April 11, 1923. At that time he had typical lesions of dermatitis repens on both hands and feet, an acute eczematous dermatitis of his penis and scrotum and behind the ears, a scaly eczema of his forehead, conjunctivitis, superficial glossitis and stomatitis. There was a profuse purulent discharge from under the foreskin, and a slight one from the urethra. He was admitted to Guy's Hospital on April 19, 1923. By that time the conjunctivitis had subsided, but the eruption had spread on to the forearms and legs in the form of pustules and eczematous lesions. The lesions on the palms and soles superficially resembled those of keratoderma blenorrhagica. The patient was kindly examined for me by Mr. V. E. Lloyd, who reported that there was acute balanitis and slight urethritis, but he thought that the urethritis was not due to the gonococcus.

Mr. E. Biddle, Chief Assistant in the Bacteriological Department, made the bacteriological investigations for me. Cultures of the skin lesions on the palms and soles, legs, forearms, penis and scrotum, all gave a pure growth of a *Staphylococcus pyogenes aureus*. The same organism was also recovered in pure culture from the urethra, from the pus exuding from under the foreskin, and from the urine. Smears from the urethral swabs and from the urinary deposit showed no gonococci.

The patient's blood serum tested against his own staphylococcus gave agglutination in dilutions of 1 in 20 and 1 in 200.

The complement-fixation tests were as follows: Gonococcus positive; autogenous staphylococcus, strong positive; stock staphylococcus, strong positive.

The Wassermann reaction is negative.

An autogenous staphylococcal vaccine was prepared and an initial dose of 25 million was followed in twenty-four hours by an acute spread of the eczematous dermatitis in the groins. His condition is now much improved and further vaccine treatment is being cautiously continued.

This case resembles those originally described by Crocker as "dermatitis repens," and Hallopeau as "acrodermite continue," although Hallopeau did not consider that his cases were identical with Crocker's. Sutton, in 1911, described three cases and gave histological descriptions, and he came to the conclusion that the cases of Crocker and Hallopeau were of the same nature. He cultivated a *Staphylococcus aureus* which he considered to be the causal organism.

#### DISCUSSION.

Dr. J. H. STOWERS confirmed Dr. Barber's diagnosis and said that this disease was of rare occurrence. He referred to a typical and severe case, which he published in the *British Journal of Dermatology* in 1896, with coloured plates, and which was exhibited at the Dermatological Society of London. The patient, a married woman, aged 67, was sent to him by Dr. Reid, of Canterbury. In this case no history of injury could be obtained, but the disease commenced in 1888 within a fortnight of the birth of her last child. The primary lesion, described as a small "gathering," occurred in the matrix of the right thumb nail. The nail became loosened and detached by inflammation. The ulceration never healed, but the left thumb, and all the fingers of both hands, became similarly involved at intervals of several months. Subsequently, the nails of all the toes became affected and the inflammatory and ulcerating process gradually spread up the whole of the hands and the distal half of each foot. The disease had been defined as a spreading dermatitis usually following injury, probably neuritic, and commencing almost exclusively on the upper extremities. The late Dr. Radcliffe Crocker, who was the first to describe this affection and who had already recorded two or three cases, saw the patient and confirmed the diagnosis. In addition to these, similar cases had been recorded by Charlton and Coward in this country, Garden, of Aberdeen, and Hallopeau, Frèche, Audry and Carle, on the Continent. The disease, always very rebellious to treatment, might ultimately yield to local remedies of an antiseptic nature but internal treatment appeared to have no beneficial effect. He (Dr. Stowers) treated his case with a lotion of permanganate of potash as suggested by Dr. Crocker, but, later, continuous application of the 5 per cent. olete of mercury appeared to produce a better result. The patient did not recover, and ultimately died of an intercurrent disease.

Dr. H. G. ADAMSON (President) said that Dr. Barber's case showed many features which suggested psoriasis: for example, the sharply-margined patches with scaly surface, and on some of the lesions being scraped the same appearance was left as in psoriasis. The patient might have psoriasis with a secondary infection.

Dr. BARBER (in reply) said that although the patient had recently developed patches which looked like psoriasis, he (the speaker) did not entertain that diagnosis, as there was an acute involvement of the mucous membranes, and the greater part of the eruption was quite unlike psoriasis.

### Case of Atrophic Dermatitis of the Hands and Feet; ? Lupus Erythematosus.

By H. W. BARBER, M.B.

B. P., AGED 51, female, was admitted under Dr. Beddard to Guy's Hospital on May 2, 1923, for breathlessness on exertion and swelling of the feet. She had suffered from breathlessness on exertion for the past fifteen years, but the swelling of the feet had only occurred during the previous ten days, and was



most marked in the evening. During the last ten years the skin of the fingers and toes has become bluish in colour and very thin. The nails have also atrophied and have almost disappeared. The changes in the fingers and toes have not been accompanied by pain or ulceration, nor is there a history of "dead" fingers such as one obtains in cases of Raynaud's disease. Dr. Beddard diagnosed senile emphysema, slight sclerosis of the mitral valve and myocardial degeneration.

I was asked to see her on account of the condition of her hands and feet. The skin of the fingers and toes is bluish red and atrophied, and the nails have almost disappeared. The appearances are not unlike those seen in lupus erythematosus of the extremities, but there has never been any eruption on the face, ears or scalp.

The Wassermann reaction is negative.

It is a point worth raising as to whether this case comes under the heading of those described on the Continent as acro-dermatitis atrophicans. The appearance of the fingers suggests to me lupus erythematosus, which I have seen with nail changes.

### Outbreak of Alopecia (Two Cases).

By H. C. SEMON, M.D.

I HAVE only had an opportunity of investigating this alleged epidemic of alopecia during the past week, but such stumps as I have examined under the microscope show no sign of being infected with tinea.

This child, aged 8, has been an inmate of the orphanage fourteen months, and entered it with typical alopecia areata. On April 27 it was noticed that she had a bald area on the right frontal region. On May 3 this next child shown was noticed to have a bald area on the left frontal region, *not* typical alopecia areata. Most of the heads in the institution were examined, and fourteen had a similar condition of atypical alopecia. The patches are not round, but of angular outline, and not completely denuded of hair, and show no typical comma-shaped stumps. We are, therefore, here dealing either with an epidemic of unknown etiology, or, as has been suggested by French writers, it is an artefact condition. Dr. Haldin Davis described an epidemic, also in a girls' asylum, in 1914; and Bowen, whose paper in the *Journal of Cutaneous Diseases* for 1915 is worthy of study, has described this peculiar condition as occurring in another girls' school.

#### DISCUSSION.

Dr. J. H. SEQUEIRA thought it most likely that these were cases of imitative artefact.

Dr. HALDIN DAVIS said he had seen an almost exactly similar epidemic in a home for children in 1914; and, several years afterwards, he concluded it must be imitative artefact. At the time he first saw it he wondered whether any of the children had cut their hair, but concluded they did not do so. But subsequently he formed the opinion that having had their attention directed to the scalp, they rubbed it with their forefinger, which soon broke hairs off. Another cause of the epidemic in that institution was, that nurses and attendants were very assiduous in searching the heads of all the children to find something, and conditions which in the ordinary way would pass unnoticed were, under these conditions, made much of.

Dr. O'DONOVAN said he had come across a parallel epidemic in Dr. Sequeira's clinic : there were five cases brought from an institution, and these at first seemed to be cases of *tinea capitis*. In the first of the five the spores were obvious ; in the others there were bald patches, and some short hairs, but several examinations failed to reveal anything. A casual remark was made by an accompanying attendant that " boys were devils," and it transpired that the boys, seeing what interest was aroused by a case of ringworm, dropped sealing-wax on one another's hair and then pulled the stumps out. In New Bond Street there was exhibited for sale a wax which was melted and applied to unwanted hair, which afterwards could be removed *en masse*. He had a case of autophytic baldness—a youth with bad asthma who, between his spasms, sat up in bed and plucked at his frontal hair. After the attacks he dyed his temporary bald patches with a solution of permanganate of potash. A parallel case of autophytic alopecia was that of a very neurotic old woman, with great trouble at home, who clipped large areas off her scalp and sought advice at the clinic for the resulting baldness.

Dr. GRAHAM LITTLE showed (1) A Case of Lupus Erythematosus ; (2) A Case for Diagnosis ; (3) A Case of Extensive Urticaria Pigmentosa Nodularis in an Infant.

### Tuberculous Lymphangitis of Skin.

By J. H. SEQUEIRA, M.D.

PATIENT, a female, aged 55, has had lupus vulgaris of the atrophic type on the face for seventeen years. Eighteen months ago she developed a sore between the right first finger and the second, and, following upon that, there developed rather rapidly an acute swelling of the forearm and arm, approaching a pseudo-elephantiasis in size. On this, which I think is the result of tuberculous lymphangitis, there have developed a large number of nodules, standing up about  $\frac{1}{8}$  in. above the surface. Histologically their structure has been found to be tuberculous. The nodules on the back of the hand and across the back of the wrist are of that type of lupus which at one time was called lupus hypertrophicus. I have not before seen so marked a case in the upper limb, but I have had several in which the lower extremity has been affected, and this has always been associated with the direct inoculation of the skin, which has been followed by lymphangitis. Improvement is very slow. This patient has not yet had any treatment ; but I hope to show her at a later date.

### Case of Acne Scrofulosorum.

By A. M. H. GRAY, C.B.E., M.D.

THIS female child, aged 6, was admitted to the Hospital for Sick Children, Great Ormond Street, under Dr. Still. She has had this eruption two years, and it has been associated with bilateral swelling of the wrists, knees and ankles. The eruption is of interest from several points of view. It consists of indolent follicular pustules of the acne scrofulosorum type, which occur chiefly on the extensor aspects of limbs, a few scattered lesions on the back, and very marked lesions on both cheeks. On the extensor aspects of the limbs there are larger lesions, varying in size of from that of a threepenny-piece to one 6 in. in diameter on the leg. She also exhibits a curious condition of scalp, in which there is, apparently, nothing very active, but which closely resembles folliculitis decalvans. The joints noted above are still swollen. There are

signs of tuberculosis at the left apex. She gives a negative Wassermann but a strongly positive von Pirquet reaction to both human and bovine tubercle. There is no recent tuberculosis in the family, nor any history of it. I think there can be no doubt that the larger patches are formed by the aggregation of the follicular pustules. In some of the small ones you can see acne scrofulosorum patches arranged round the main lesions. There is also no evidence of lupus nodules in these larger patches. Sections showed a granuloma of the tuberculous type, but tubercle bacilli could not be found. The lesions on the face are, I believe, a true example of Barthélemy's "acnitis," and it will be observed that they bear no resemblance to Crocker's "acne agminata" nor to Boeck's "miliary lupoid." The scalp condition is apparently of the same nature as the rest of the eruption and it so closely resembles a "folliculitis decalvans" as to raise the question as to whether this latter condition may not possess a possible tuberculous ætiology. I have not seen this suggestion made.

#### DISCUSSION.

Dr. S. E. DORE thought the case important; the association of what appeared to be folliculitis decalvans with tuberculides seemed to be unique. He asked whether folliculitis decalvans ever occurred in children. He thought the scalp condition was part of the tuberculosis due to the same condition as the eruption on the skin.

Dr. J. H. SEQUEIRA said he hoped the case would again be shown before the Section as he believed that this scalp condition associated with tuberculides was unique.

## Section of Dermatology.

President—Dr. H. G. ADAMSON.

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### Case for Diagnosis ; ? Papulonecrotic Tuberculides.

By E. G. GRAHAM LITTLE, M.D.

THE patient is a girl, aged 16, over-fat, with a chilblain circulation and an unhealthy colour. I have suggested as a tentative diagnosis, papulonecrotic tuberculide of an acneiform type. The history is very curious: She had four attacks of measles in six years, the last attack being in 1912. Twice in 1915 and 1916 she had an eruption diagnosed as scarlet fever and she had an attack of jaundice eighteen months ago. The present eruption appeared about a fortnight ago; it came on fairly acutely, but she seems to have had similar attacks previously, though the history in this respect is rather indefinite. The eruption consists principally of hard shotty papules with a minute central pustule. It is thickly distributed on the forearms, less thickly on the dorsum of the hands. There is no itching. On both upper arms just below the shoulder there are some areas of grouped follicular papules which are almost certainly examples of lichen scrofulosorum. The patient's father tells me that up to two years ago she was very thin and anæmic and has become very fat only within the last few months. This plumpness I take to be the *faux embonpoint* which the late Dr. Pringle used to point out as being typically tuberculous. There do not appear to be any other symptoms of tubercle at the present time.

### Xantho-erythrodermia Perstans (Crocker), *Parapsoriasis en plaques* (*Erythrodermie pityriasique en plaques disséminées*, Brocq).<sup>1</sup>

By H. C. SEMON, M.D.

IN his article in the *British Journal of Dermatology* of 1905,<sup>2</sup> Dr. Crocker describes a disease which closely conforms to the case here exhibited. He also states that the name xantho-erythrodermia perstans was suggested by Dr. Pernet. The same condition is described, under the name of "parapsoriasis," by Brocq. It is a slightly scaly reddish-yellow, irregularly distributed collection of patches, mostly on the thighs, although there are a few lesions on the arms and trunk. Subjective sensations are so slight that the patient, a young man, himself is not at all sure when he first noticed the eruption—he thinks about

<sup>1</sup> Brocq, "Dermatologie pratique," 1907, ii, p. 367.

<sup>2</sup> *Brit. Journ. Derm.*, 1905, xvii, pp. 119-134.

a year ago. Throughout the two months during which I have been treating him with various ointments and the Kromayer lamp, there has not been the slightest change either in number or appearance of the lesions. The eruption corresponds intimately to the seven points postulated by Brocq: (1) Almost complete absence of pruritus; (2) very slow evolution; (3) scattered, circumscribed, sharply defined patches of varying size (2 to 6 cm.) and situated mainly on the thighs and trunk; (4) a pinkish coloration; (5) absence of infiltration; (6) slight branny desquamation; (7) pronounced resistance to treatment of all kinds.

### Two Cases of Neurofibromatosis.

By S. E. DORE, M.D.

THESE are two striking examples of extensive multiple tumours of the skin. One, that of a woman, aged 44, is a typical case of molluscum fibrosum. She has had the condition seven years. There are numerous soft flat and protuberant tumours scattered over the body, some with blue discoloration, and there are also some patches of pigmentation. The other patient, a woman aged 40, has had multiple pigmented growths from birth; she says they have increased in number, and some have increased in size. There is also a large raised pigmented warty area of skin in the sacral region. Many of the small prominent tumours are free from pigment and I have classed both these cases under the generic term of neurofibromatosis, but several members who have seen the second case consider it to be one of pigmented moles.

### Parakeratosis Variegata in a Man, aged 40.

By S. E. DORE, M.D.

THIS is a case of what I believe to be parakeratosis variegata. The patient has had the eruption for twelve years; it began with three small patches on the trunk, and there has been a gradual increase in the number and size of the patches. There is considerable itching, especially when the patient becomes hot and sweats. For many years this man has driven a steam engine and it is possible that exposure to heat has aggravated the condition. There are large circular patches of dermatitis scattered over his body of a deep red or brown colour, some of them having a retiform or stippled appearance. The recent patches are covered with fine scales and somewhat resemble psoriasis.

A patient, suffering from this disease, whom I showed here in November, 1922, who was originally shown by Dr. Wells Patterson at the Newcastle meeting of the British Medical Association, and was subsequently under the care of Dr. Cranston Low in Edinburgh, developed an indurated mahogany-coloured scaly skin and has recently died at the London Hospital from leukaemia. I remember the late Dr. Pringle asking whether anything was known as to the further history of these cases of parakeratosis variegata. The case I refer to changed completely, as regards his cutaneous condition, from parapsoriasis to a generalized scaly pigmented erythrodermia, and it would appear that the latter was related to the leukaemia rather than the former.

## DISCUSSION.

Dr. H. MACCORMAC said that mycosis fungoides might be suggested as an alternative diagnosis. It could be put to the test in two ways: by microscopic examination and by the result of the application of X-rays—if it were mycosis fungoides, one would expect a rapid clearing up after X-rays. The long duration did not exclude mycosis fungoides as in some cases the premycotic stage had existed for years. He did not know whether the infiltrated character of the lesions allowed the condition to be included under the term "parapsoriasis." The considerable degree of itching was of importance in the diagnosis.

Dr. W. J. O'DONOVAN confirmed the statement of Dr. Dore that a patient who was considered to have parakeratosis variegata had died in the London Hospital, and a post-mortem examination had been made. He was taken in as a case of bronze diabetes, but when his blood came to be examined the condition was found to be that of "homme rouge." This man, however, had a spleen which weighed 5 lb.

Dr. GARDINER said that at first he had thought this was a case of mycosis fungoides in the premycotic stage. There were several areas on the back which were distinctly atrophied, and there was a marked thickening on the thigh. There was also itching, and he agreed that premycotic itching might exist for years before nodules appeared. In most of the cases of parakeratosis that he had seen, the areas chiefly involved had been the forearms, and there had been definite linear markings; the appearance was different from that in this case.

Dr. F. PARKES WEBER said that in this case an early leukæmic condition could be practically excluded, as there was no obvious infiltration of the skin, but a blood-count should be made. He thought there was danger of misunderstanding regarding the case referred to by Dr. O'Donovan; he had recently seen that case in the London Hospital (May 30), and it was then very unlike the present case of parakeratosis. Practically the whole skin was infiltrated, and the patient (a man, aged 60), had a definite leukæmic change in the blood. He had heard that the case had been claimed to be one of lymphoblastic erythrodermia, and he told Dr. Robert Hutchison, who kindly allowed him to see the case, that if the liver could be examined it would probably be found to show lymphocytic (leukæmic) infiltration. Probably all the cases published by Dr. Sequeira and Dr. Panton as lymphoblastic erythrodermia were leukæmic skin cases of the class described by Kaposi under the term "lymphoderma perniciosum."

Dr. GRAHAM LITTLE said that he supported Dr. Dore's view. He did not think this was a case of mycosis fungoides, but one of parapsoriasis. With regard to the case of erythrodermia mentioned by Dr. O'Donovan, it was a little unfortunate that the term "homme rouge," already allocated to describe the terminal state of mycosis fungoides, should be used in the description of any other type of disease.

### Extensive Linear Nævus in a Man, aged 54.

By S. E. DORE, M.D.

THIS is an extensive linear nævus, and somewhat resembles the case of a girl described by Dr. Stowers many years ago. In this patient the nævus is on both sides of the face, but is on the right side of the body only. On the face there are masses of closely aggregated, deeply pigmented papillomatous lesions. There is a long streak in the central line of the abdomen, a large patch in the right groin, and another streak extending down to the foot. Some of the growths were removed surgically from the chin, but they have recurred to a certain extent. I do not think it is worth while attempting anything in the way of active treatment.

Dr. J. H. STOWERS said that this case corresponded, in many particulars, with the one he (Dr. Stowers) had shown before the Dermatological Society of Great Britain and Ireland, many years ago, full details of which were published with illustrations.<sup>1</sup> He agreed with Dr. Dore that surgical treatment alone was likely to be of service to the patient.

### Case of Mycosis Fungoides.

By HENRY MACCORMAC, C.B.E., M.D.

THE patient, a male, aged 37, states that about four years ago, he noticed some small lumps on the right side of the left ankle. About two years later the local practitioner who first saw the condition, considering it to be syphilitic, sent the patient to hospital, where a diagnosis of mycosis fungoides was made. The disease by this time had spread considerably, the patient being extensively affected on the trunk and limbs. He was treated with X-rays, and following that treatment the eruption completely disappeared. Three subsequent relapses have occurred, the condition as seen at present being more extensive and severe than any of the others. It was formerly possible to remove the eruption with comparatively small doses of X-rays, but the lesions, especially the tumour formations which are represented here and there, are now much more resistant, requiring a full pastille dose. Repeated blood-counts have been made, but despite the very extensive application of X-rays to the skin surface, no degree of anæmia has as yet become evident. There is a slight leucocytosis.

### Dermatitis Artefacta.

By HENRY MACCORMAC, C.B.E., M.D.

THE patient is a single woman, aged 24, a cook by occupation. Eleven months ago she burned both legs with hot fat, but whereas the lesions on the left leg healed rapidly, those on right persisted. When the patient was seen at hospital (February, 1923), there was a large erythematous patch on the right leg, with superficial scar-formation, peculiarly translucent and showing numerous superficial vessels just beneath the surface. The lower margin was marked by a well developed erythematous border which was slightly eroded. No progress was made while the patient remained an out-patient, but after admission to the ward, and after the affected part had been enclosed in plaster of Paris, complete healing took place, the scar alone marking the site of the eruption.

### Case of Morphœa associated with Vitiligo.

By H. W. BARBER, M.B.

A. L., AGED 54, male, dock labourer. The patient has had pruritus scroti for about thirty years, and came to me in May of this year on account of this symptom. On examination, apart from lichenification of the scrotum, vitiligo of the penis and groins was observed, also a large area of morphœa with lilac border in the mid-line of the lower part of the back and symmetrical patches

<sup>1</sup> *Brit. Journ. Derm.*, 1908, xx, p. 1.



of morphœa on both sides of the lower abdomen and groins. As these patches did not attract the patient's notice he does not know how long they have been present. Physical examination revealed no abnormality except very severe oral sepsis. Wassermann reaction negative.

Mr. Bulleid has investigated the mouth for me. Apart from gingivitis, pyorrhœa with pockets, radiograms revealed extensive apical infection of the teeth with marked bony changes in the alveolus. Several teeth have been removed, and a pure growth of *Streptococcus longus* was recovered from the apices. This organism was agglutinated by the patient's serum in dilutions of 1 in 20 and 1 in 200.

Streptococci obtained from the tonsils were not agglutinated. Cultivations of the faeces (three specimens) gave only *Bacillus coli* and a few colonies of a *Streptococcus faecalis*, which were not agglutinated by the patient's serum.

Since being in hospital the patches of morphœa have become much less evident.

On February 28, 1908, Dr. Adamson showed a case of scleroderma and leucoderma combined in a patient, a girl, aged 16. "On the neck and chin on the left side there was a large irregular area of leucoderma with a margin of deeper pigmentation and some finger-nail-sized pigment macules over the white area. Occupying part of the same area were three elongated patches of scleroderma. Apart from the interest of the unusual association of leucoderma and scleroderma was the fact that the distribution corresponded very closely with the sensory area of the second and third posterior cervical roots. Towards the chin, however, it overlapped this area and passed on to that of the third division of the fifth cranial."<sup>1</sup>

Dr. F. PARKES WEBER regarded the present case as a genuine example of the association of vitiligo and the morphœa form of scleroderma; but in some other cases the supposed vitiligo lesions were in reality only part of the scleroderma.

### Generalized Scleroderma with Subcutaneous Nodules.

By A. M. H. GRAY, C.B.E., M.D.

THE patient is a young woman, aged 26. She has diffuse scleroderma with a four years' history. It started as the hypertrophic œdematous form, which gradually subsided, and she now presents the typical atrophic form. It is apparently getting considerably better. The interest of the case lies in the fact that during the last year she has noticed numerous subcutaneous nodules. They are limited to certain parts of the body, and appear to be attached to the tendon sheaths, capsules of joints, and periosteum. On the tendon sheaths at the backs of the hands are many of them, closely arranged in lines; their size is a little less than that of a lentil. There are many in the region of the tendons of the hamstring muscles, and on the dorsum of the foot. In addition there are larger fibrous nodules attached to the periosteum in the neighbourhood of joints and along the iliac crests. The largest nodules are on the back of the scapulæ, and attached to the spinous processes of the dorsal and lumbar vertebræ. There are a few scattered about on either side of the spine, apparently not attached to periosteum nor to the deep fascia. I removed two of them from the dorsal region, and they were not fixed down to the deep fascia, but were free in the subcutaneous tissue.

<sup>1</sup> *Proceedings*, 1908, i (Sect. Derm.), p. 43.

Subcutaneous nodules are not very uncommon in sclerodermia; and Crocker believed that they would be found to be common if they were looked for. But, apart from calcareous nodules, there have been very few references to nodules not of a calcareous nature. I have had some of the nodules in this case X-rayed, and they are not calcareous; I have also examined sections, and these do not show any deposit of chalk in the tissues. Microscopically, the fibrous tissue of the nodules has apparently undergone hyaline degeneration, and in addition there is a very closely packed round-cell infiltration round the vessels at the periphery of the nodule itself.

Dr. F. PARKES WEBER said these nodules reminded him of the "rheumatic nodules" sometimes seen in young adults; and Radcliffe Crocker had referred to the occasional occurrence of "subcutaneous nodules of the rheumatic type" in association with sclerodermia. In the present case the distribution of the nodules about the scapulæ and iliac crests resembled that of rheumatic nodules. He (Dr. Weber) had seen the case in August, 1922, and had then specially noted the beaded (nodular) condition of the tendons or tendon-sheaths at the back of the hands.

### Case of Acne Varioliformis.

By W. KNOWSLEY SIBLEY, M.D.

S. B., AN unmarried woman, aged 26, was sent to me by Dr. E. J. Williams for a skin condition from which she had been suffering for over four years. The lesions were chiefly aggregated at the hair margin of the forehead, but extended slightly down on the face in front of the ears. There were acneiform papules leaving large well-marked scars. A group of similar lesions had been present on the upper dorsal region between the scapulæ, where some dozen very large varioliform-looking scars are present.

### Case for Diagnosis.

By J. E. M. WIGLEY, M.B.

THIS little girl is said to have had a recurrent blister eruption every year since she was vaccinated at 6 weeks of age; she is now aged 12. The blisters and pustules are situated chiefly on all four extremities, and there are a few on the trunk. The eruption appears in the spring, stays during the summer, and disappears in the winter. This year, for the first time, pustules have occurred; previously there had been only blisters. Itching is said not to be very severe, but more marked at night. Cultures from the clear blisters are sterile, but from the pustules we obtained a long chain streptococcus in pure culture. The Wassermann reaction is negative. I think it may be a case of dermatitis herpetiformis.

### Case of Lichen Spinulosus.

By J. E. M. WIGLEY, M.B.

PATIENT, a child, aged  $4\frac{1}{2}$ , apparently healthy, has a local condition which I think is lichen spinulosus, associated with a mild degree of xerodermia, and I think there is some evidence of early achondroplasia. There is marked lordosis, and shortening of the upper portions of its limbs. The Wassermann reaction is negative. Has that anything to do with the ætiology of the other condition?

## A New Method of treating Skin Diseases.

By Sir G. ARCHDALL REID, K.B.E., M.B.

(Introduced by H. G. ADAMSON, M.D., President.)

IN treating the cases which will now be shown, I have been using salicylic acid in great strength—50 per cent. by weight in vaseline and about 12 per cent. in collodium. My idea was that, when weak preparations of the acid were used neither the pathogenetic organisms nor the surface tissues were destroyed; as a consequence, absorption, inflammation, and perhaps poisoning resulted; but, if the acid were used in great strength, the surface tissue and the contained organisms would quickly be killed, and a dead layer formed which would protect against absorption. Apparently this is what happens. On healthy skin the layer of tissue destroyed by this treatment is no thicker than tissue paper. On diseased surfaces little besides the affected tissue perishes. The whole thickness of the epidermis is not destroyed, except perhaps in minute, punctiform, diseased areas. The 50 per cent. ointment may be left on for days, and in a day or two after its removal a smooth, unbroken skin is generally to be seen. I tried this ointment on myself first, and then, very cautiously, on patients. I have never had any ill results. There has never been undue pain, or much inflammation, or so much as a suggestion of poisoning. Now I use it boldly over large areas.

The following notes briefly indicate the results of treatment in certain skin diseases:—

*Lupus.*—In the treatment of lupus by this method swift improvement results—especially in the case of lupus vulgaris. The cosmetic results are excellent. But I have never yet achieved complete cure. Of two female patients now shown, one will be seen with a scar on her nose. A fortnight ago she had an open sore and the nostril was being eroded. I have brought her to show that salicylic acid, used in strength, heals, but does not destroy. The other lady, whose condition has greatly improved, had extensive lupus erythematosus of many years' standing over one eyebrow and on a large area of scalp.

*Scabies.*—I had one case of this affection in which I treated the patient with the ointment, and recovery very rapidly followed.

*Ringworm.*—There are several cases for demonstration, two or three of which are only half cured, and I have brought the patients to show how little irritation is caused by the ointment, rubbed in for ten minutes twice a day. Dr. Victor Blake, who is in charge of the Portsmouth school clinic and who has tried the treatment, has also brought some cases of ringworm, and these he will describe to you himself.

*Dermatitis.*—But the most striking results are seen in cases of dermatitis, erythematous, vesicular, papular, scaly, acute, chronic, some very extensive. The results of the treatment are summarized in the following cases shown:—

*Case I.*—This is the case of a young girl, aged 16, who, a month ago, developed acute vesicular dermatitis on the side of her face and neck. Now you can see only faint traces of past trouble. Her fresh complexion indicates how little injury is caused by the strong ointment.

*Case II.*—That of a young woman who two months ago suffered from very acute vesicular dermatitis over one side of her face and neck and over both arms and hands. In less than a week there was small trace of disease left. She suffered great

pain on the first day of treatment, less on the second, still less on the third, and on the fourth she was without pain. This is a common experience.

*Case III.*—Patient, a man, is a baker. He had been under private treatment and as he was getting no relief he was advised to attend the hospital. A month ago he came to me with his forearms a mass of scabs. In a few days he had greatly improved. But when he was about to return to work he suddenly developed acute dermatitis of both legs below the knees—so acute that the legs became oedematous. That was a fortnight ago; but beyond some redness and roughness of the skin he has little trace of the trouble now.

*Case IV.*—Patient, a female, who had a papular vegetative dermatitis of the arms and hands for fourteen years. In a week she had not a trace of it.

*Case V.*—Patient, a female, had papular dermatitis of both forearms, and scaly dermatitis from the middle of her thighs to her ankles for eight years before she came to me about two months ago. She, too, now has hardly a trace of the trouble remaining. But unfortunately she shows great tendency to relapse when she discontinues the ointment.

Dr. VICTOR J. BLAKE said it was only within the last month or six weeks that he had tried the treatment, but in cases of acute dermatitis, eczema, &c., he had had one or two remarkable results. One was a child whom he had hoped to bring to the meeting; she was aged 6 years, and had had trouble since her second year. All kinds of treatment had been tried, but four days after commencing Sir Archdall Reid's treatment the condition was well, and that was a month ago. In two other cases the eczema healed in two or three days. When the surface was raw, he felt some compunction about applying this strong treatment, but he did so, and in four days the condition had cleared up, and there had not been any recurrence. With regard to ringworm cases, he was somewhat of a sceptic. Having many ringworm cases to deal with, he had given up treating them by drugs, and had relied upon X-rays. However, since using the method now under discussion he had had several cases in which there was very rapid improvement. He was never satisfied with a case of ringworm unless he could find the scalp free from infected hairs, and the microscope showing no spores; but in the cases they had submitted to this treatment there seemed microscopically to be a marked degeneration in the spores. A small child under his care was given the treatment a month ago, and its ringworm was now absolutely cured. Equal parts by weight of vaseline and salicylic acid were used, and the efficacy of the ointment thus compounded was very much greater than that of all the ointments which had been tried for ringworm.<sup>1</sup>

<sup>1</sup> Owing to the lateness of the hour no opportunity was given to Members of the Section to discuss the cases shown.

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## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

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### Cardiac Diagnosis—a Survey of the Development of Physical Methods :

#### PRESIDENT'S ADDRESS.

By ROBERT KNOX, M.D.

DISEASES of the heart form a large proportion of the most serious disorders that afflict mankind, both in youth and in adult life. From the earliest time attention has been directed to methods which would enable the physician to determine the normal action and function of the heart, and which would enable him to diagnose departures from the normal.

In the early days of medicine the absence of correct anatomical, physiological, and pathological knowledge prevented diseases of the heart from being recognized during life, and almost entirely precluded them from becoming the object of medical treatment. The first great advance was made in 1623, when Harvey published his discovery of the circulation of the blood, and its dependence on the heart as the central organ of the circulatory system. From this time onwards derangements of the circulation began to be recognized as signs of disease of that central organ. The earliest evidences of this important step in the diagnosis of these diseases are to be found in the instructive records contained in the writings of the great pathologists of the eighteenth century, of whom the chief in this respect are Lancisi, Senac, and Meckel.

There is, however, a limit to the information obtainable by such observations; and in these early times it was very quickly reached when men had only their unaided senses upon which to depend, and but little clinical experience to guide them. It is not surprising, therefore, that after the first great step was taken there was little further progress made, and medicine had to wait till the beginning of another century, when Corvisart (1806) first made practical use of Auenbrügger's invention (1761) of percussion to determine the size of the heart, enlargements of that organ bulking largely in those days as a substantive disease. Corvisart was also in the habit of listening to the sounds made by the heart, and he may, therefore, be regarded as the first practical exponent of modern methods of physical exploration, though scarcely as their founder. Just as Auenbrügger invented percussion so Laënnec (1819) was

undoubtedly the first to make a scientific application of mediate auscultation to the diagnosis of diseases of the chest by the introduction of the stethoscope, and it is to him that we trace all the benefits derived from this method of inquiry, though it is to Bouillard (1824) that we are chiefly indebted for its extension to the diagnosis of diseases of the heart, while it is to the clinical inference, and above all to the practical experiments of James Hope (1832) that we owe much of that precision to which we have now attained in the diagnosis of these diseases from abnormalities in the sounds produced during the cardiac movements.

In Italy the tradition of the great anatomists and physiologists produced a series of accurate observers and practitioners; amongst the first of these were Antonio M. Valsalva (1688-1723), still better known as an anatomist, the author of a classical work on the diseases of the heart and aneurysms, Ippolite Francesco Albertini (1662-1738), whose researches in the same class of disease were no less important, and Leopold Auenbrügger (1722-1809), the inventor of the method of recognizing diseases of the heart by percussion.

Auenbrügger's method was that of direct percussion with the tips of the fingers, not that which is now used of mediate percussion with the intervention of a finger or pleximeter, but the results of his method were the same, and its value nearly as great. Auenbrügger's great work, the "*Inventum Novum*," was published in 1761. Surpassed, but not eclipsed, by the still more important art of auscultation introduced by Laënnec, this simple and purely mechanical invention, it is hardly too much to say, has had an influence upon the development of modern medicine greater than all the systems evolved by the most brilliant intellects of the eighteenth century.

John Nicholas Corvisart (1755-1821) translated and introduced into France Auenbrügger's work on percussion. He made some improvements in the method, but the only real advance was the introduction of mediate percussion by Ferri in 1828. The discovery had yet, however, to be completed by that of auscultation, or the listening to sounds produced in the chest by breathing, the movements of the heart, &c. The combination of these methods constitutes what is now known as physical diagnosis.

René Théodore Hyacinthe Laënnec was the inventor of this most important, perhaps, of all methods of medical research. It was definitely expounded in an almost complete form in his work, "*Traité de l'Auscultation Médiate*," published in 1819.

John Forbes in 1824 and William Stokes of Dublin, pupils of Laënnec, introduced the stethoscope into this country. Forbes translated the works of Laënnec and Auenbrügger, and an entire revolution was soon effected in the knowledge of diseases of the chest.<sup>1</sup>

The works referred to by no means exhaust the literature of the subject during the past ages, but each of these marks an era, and by their means we may readily trace the progress of cardiac diagnosis during the one hundred and thirty years intervening between Lancisi and Stokes.

Since then the literature has become so unusually copious and exhaustive that it would occupy too much space merely to enumerate the authors' names.

<sup>1</sup> The record so briefly referred to is from the work of Dr. G. W. Balfour, an Edinburgh physician, who did much to advance the cardiological diagnosis in his time. To continue this record, and bring it up to date so far as mechanical methods are concerned, will be the object of the present address.

The purpose of the present paper is to emphasize the importance of the later and by no means least important of the developments in cardiac diagnosis. Reference has been made to the purely historical events in diagnosis to enable us to realize how gradual has been the development, and how dependent the later and more exhaustive works and methods have been upon the genius of the earliest pioneers; for instance, without Harvey's brilliant and original work upon the circulation of the blood the progress of cardiological technique might have been indefinitely delayed.

As in former so in later times, the progress made in the diagnosis of diseases of the heart has been almost entirely based upon physics and physiology. The heart is an organ so situated and so connected as to render it singularly amenable to these methods of investigation. Resting as it does upon the diaphragm and projecting against the anterior chest wall it is practically within reach, and therefore favourably placed for the observations required in its investigation. These, as already indicated, are the valuable methods of inspection, palpation, percussion, and auscultation. Great experience is required when these are relied upon for a diagnosis, and they must always retain the chief place in the estimation of the skilled physician. But there are now a number of other aids to diagnosis which are of recognized value in the investigations of the actions of the heart, and the departure from the normal to which it is liable, and as these are largely physical in character it will be well to consider them briefly.

Physics has played such an important part in the development of methods of diagnosis that it is essential for the future progress of this branch of medical work that the clinician should not only have the advantage of the best technical skill in the development of instruments, but be familiar with the technical details of the apparatus provided. In order to comply with these requisites he must have an intimate knowledge of physiology and physics and electro-technique. The use of electricity as instanced, for example, by the electro-cardiograph, has already rendered important service in the development, while the gradual evolution of the apparatus has necessitated the employment of the highest inventive power and technical skill. Such development is a standing testimony to what can be accomplished in the way of exact instrumentation.

The modern physician enjoys many advantages denied to his predecessors. It will be our object to deal with the later developments of instrumentation, and to indicate in what way these advances have led to improvements in cardiological technique.

The spirit of investigation stimulated by the discovery of the circulation of the blood was soon at work in various directions. The great physiologists of the generation following Harvey put the physics of the circulation of the blood experimentally on a sound basis; investigations of, and experiments on, blood-pressure carried out in animals were evidence of an intelligent appreciation of the physical principle underlying the circulation of the blood. The heart is regarded as a muscular pump, the function of which is to circulate the blood through the vessels of the body. Many ingenious pieces of apparatus can be traced to the pioneer workers by whose names they are as a rule known. These observations combined with extensive clinical experience led to the accumulation of an immense amount of data of great value.

The next important step in the development of the technique was the introduction of Einthoven's string galvanometer, an instrument of precision in the investigation of cardiac conditions. It was an improvement on all other

clinical methods because it afforded an opportunity of studying the action of the heart in health and disease, by the employment of electrical apparatus capable of recording the activity of the heart. A great deal of very valuable information may be obtained by its use.

#### THE ELECTROCARDIOGRAPH.<sup>1</sup>

The use of this instrument in its present form has been rendered possible by the researches of Waller and Einthoven. So long ago as 1865 Köl liker and Müller demonstrated variations in the electrical condition of the exposed and active frog's heart; in the human subject it is manifestly impossible to make electrical contact with the living heart muscle for experimental purposes, but to an English physiologist, the late A. D. Waller, is due the conception that although the heart muscle is not directly accessible in the living subject, yet the organ is surrounded by masses of conducting tissue, by which variations in the heart's electrical condition may manifest themselves upon the surface of the body. He held it therefore to be possible, by the application of suitable electrodes to the moistened skin, to register changes in the electrical potential of the heart muscle itself. This was demonstrated to be the case by Waller, and he worked out a scheme of potential differences obtainable upon the surface of the body as the result of the differences obtainable in the heart muscle itself. He figured the body as divided into two parts by an oblique plane—the part above and to the right assuming the potential of the base of the heart, while that below and to the left assumed that of the heart's apex.

This exemplifies the broad fundamental principle of Waller's discovery: in practice various modifications have to be introduced.

At the time of the publication of Waller's results (1889) the most sensitive instrument available for recording minute differences of potential was the papillary electrometer. This instrument, however, possesses the defect of becoming relatively sluggish in response when made extremely sensitive to minute potential differences. On the other hand, acceleration of response can only be obtained at a sacrifice of sensibility.

These difficulties were overcome by Einthoven, of Leyden, who furnished an ideal instrument for the purpose in his "string galvanometer." This apparatus consists essentially of an exceedingly fine conducting fibre, placed between the poles of a powerful electro-magnet. The fibre, which is made of platinum, or of silvered quartz, has a diameter of only 0.001 to 0.003 of a millimeter, and is screwed at both ends so that it is free to vibrate in the same way as a violin string, from which, indeed, the apparatus derives its name.

Now it is known that a wire carrying an electric current when placed in a magnetic field undergoes deflection in a direction perpendicular to the magnetic line of force. The direction and extent of the deflection of the conducting fibre will depend upon the magnitude and direction of the current which it conveys when the magnetic field remains constant. Hence, if the terminals connected with the two ends of the galvanometer fibre are brought into contact with the moistened skin by means of suitable electrodes the fibre will execute movements which for their extent and direction depend upon the variations of potential obtaining upon the body surface. For convenience of recording the movements the image of the thread is projected upon a moving photographic plate or film. It is interesting to observe that as late as 1909 Waller himself

<sup>1</sup> From Dr. Colwell's "History of Electrotherapeutics."

regarded the apparatus, although of physiological interest, as probably not capable of giving information of any special value when used as an instrument of clinical diagnosis.

The late Professor Wertheim Salamonson, of Amsterdam, in the course of his investigations on interrupted currents found it advisable somewhat to modify Einthoven's string galvanometer. Adopting some modification in its principle

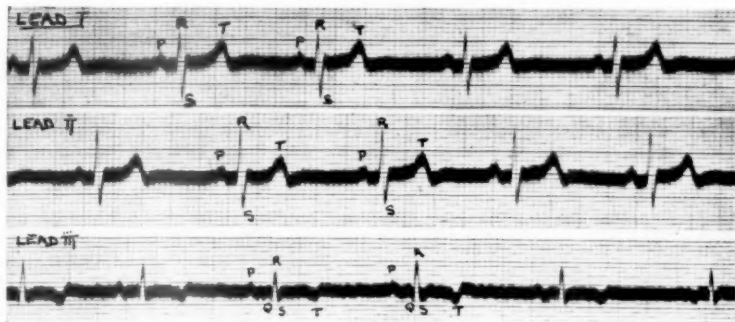


FIG. 1.—Normal gram: Shows Leads i, ii, and iii, of a normal heart. Ordinates 1 mm. = 1/10,000 volt; abscisse, fifths and twenty-fifths of a second for thick and thin lines respectively.

Characters of normal gram: Auricular complex = P, deflection, should be upright in all three leads. Ventricular complex = Q, R, S, and T, deflections. Q—directed downwards—is inconstant. R—directed upwards—usually the most prominent summit. Amplitude variable. S—directed downwards—very variable amplitude. T—should always be directed upwards in Lead ii. May be inverted in Lead i, often inverted in Lead iii.

Time factors: P, R, interval (beginning of P, to beginning of R), marks auriculo-ventricular conduction time. Normal average 0.13 to 0.17 sec. If over 0.2 sec. some heart-block present. Q, R, S, deflections: Normal duration under 0.1 sec. If longer there is delay in spread of excitation wave in ventricle. T, wave: The end of T wave corresponds with second sound, and marks the end of ventricular systole.—(Dr. Harold Wiltshire.)

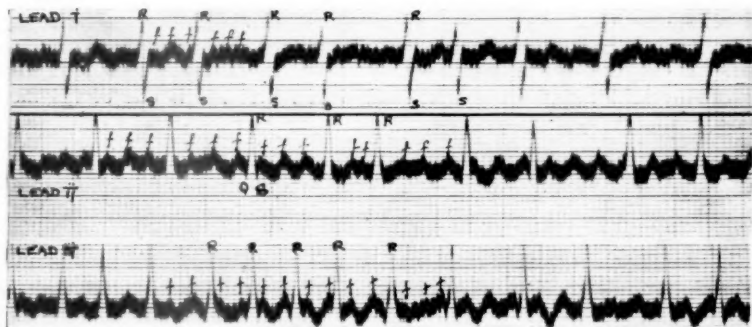


FIG. 2.—Auricular fibrillation: The ventricle is beating very rapidly (150 per min.), but it shows very marked irregularity. The normal P wave (auricular complex) is absent, and is replaced by a continuous rapid series of fine irregular oscillations, marked *f*.—(Dr. Harold Wiltshire.)

by Professor Place, he introduced the instrument named the Place-Wertheim Salamonson galvanometer. In his cardiac work at the University of Amsterdam he used this instrument extensively. By its aid he demonstrated the current derived from the foetal heart of the chicken, and propounded the hypothesis that the sharp tops of the curves were dependent upon the striated muscle fibres (figs 1, 2).

#### X-RAYS IN CARDIOLOGICAL DIAGNOSIS.

The discovery of the X-rays by Röntgen marks another important step in the development of technical apparatus for the investigation of the heart and the great vessels. Soon after the announcement of his discovery in 1895, screen examinations of the chest revealed the heart in full activity. It is not recorded, so far as we know, who was the first to examine the chest under the X-rays: probably a number of observers did so at the same time. Now for the first time it was possible actually to see through a patient, and to observe the action of the heart and the great vessels. This development marks a great step, almost an epoch-making step, for with the improvement in apparatus it soon became possible to make observations on the heart in all the phases of its activity. In quick succession were developed (1) the screen method of observation; (2) radiographic records; (3) orthodiagraphy; (4) teleröntgenography; (5) instantaneous radiography, showing the heart in various phases of its activity; and (6) the slit diaphragm records with the moving plate of Gotch and Rosenthal and later of Crane. More recently Vaquez and Bordet, of Paris, have elaborated a technique for the investigation of cardiac enlargement. Morison and White in this country described an adaptation of the method.

##### (1) *Screen Observation.*

On the screen examination alone a diagnosis can often be established. The mere visualization of the heart shadow and its movements is comparatively easy, but if more accurate records are required then a modification of the methods must be used. For accurate measurements of the outline of the heart the usual distance of about 24 in. is inadequate, because of the distortion arising from the short distance between the focus spot of the tube and the screen. At a distance of two metres this distortion is, for practical purposes, eliminated, and a true outline of the cardiac shadow is shown.

##### (2) *Radioscopy.*

The investigation of the cardiac shadow by radioscopy is one of the most valuable aids to diagnosis; it requires, however, very accurate knowledge of the normal and its variations, quickness of observation, and considerable deductive power. The movements of the heart itself are complicated by changes in its shape and size due to respiratory movements; these, while they are very confusing, can be eliminated when the observer is experienced. All variations in the movements of the heart can be recorded accurately. A further development of the screen method will be reached when photography of the screen picture becomes a practical accomplishment. At present what has been done is not satisfactory; a development of the method combined with the cinematographic projection of the film will be a great achievement, and will allow the clinician to study the movements with great advantage. The projection of the image by means of the slow movement should allow of a careful analysis of the normal and its variations.



(3) *Orthodiagraphy.*

The earliest apparatus for orthodiagraphy was introduced by Moritz, who used it in the horizontal position. Levy-Dorn is responsible for that used in the perpendicular position; both of these instruments were, for the period at which they made their appearance, far in advance of any other existing apparatus.

Groedel, in an article published in the *Interstate Medical Journal*, of June, 1911, gives the following valuable points in favour of orthodiagraphy.

He states that X-rays may be used for four purposes when examining the heart: (1) Observing the action of the heart; (2) determining the position of the heart; (3) ascertaining the form of the heart; (4) defining the size of the heart. Orthodiagraphy will probably continue in use in the future where examinations of the heart by X-rays are carried out regularly. The great advantage of orthodiagraphy is the employment of only one small sheaf of X-rays, which is carried round the heart, thus showing its outline.

The first apparatus, which could be used for horizontal drawings only, was that of Moritz. For perpendicular examinations that of Levy-Dorn was a very practical construction. Groedel succeeded in modifying this considerably some years ago. Perpendicular orthodiagraphy, being much more practical than horizontal, has now come into general use. If examinations are always made in the sitting posture, the results are, for the purpose of comparison, in every respect indisputable. The special value of the orthodiagram is to be found in the exact reproduction of the form of the heart and its details; the measurements are only of secondary importance, for in spite of all its exactness the orthodiagram gives only approximate and not absolute measurements. For the measurement of the orthodiagram it is sufficient to keep to the few dimensions that can really be delimited with accuracy. These are: (1) The greatest distance between the right and the left margins of the heart and the middle of the sternum; (2) the median distance to the right and to the left, which together give the transverse dimension; and (3) the long diameter, i.e., the greatest distance of the heart apex to the point of the division between the two right curves.

By the examination of a large number of persons with healthy hearts for these dimensions a list of average figures has been drawn up. This was done for horizontal orthodiagraphy by Dietlon, for perpendicular orthodiagraphy (sitting) by Groedel, and for children's hearts by Veith. In this way it is now possible to detect even relatively slight deviations of the heart-measurements from the normal conditions. It is not, however, possible to measure the size of the heart to within a few millimetres. In this respect the orthodiagram is not so reliable as the quick exposure negative taken at 2½ metres distance.

The numerous examinations of healthy hearts have shown, even when the size and weight of the body and the age of the individual are taken into account, that there are still great variations in the average figure.

(4) *Teleröntgenography.*

The method which bears this cumbersome name is one of considerable value. For accurate work a very powerful outfit is required because of the distance between the tube and the sensitive film. Two metres is regarded as a distance at which distortion is negligible. If the work is done at a shorter distance allowance must be made for the distortion. Salmond (*Archives of Radiology*, 1919, xxiv, p. 117) published a number of experiments

made with a lead plate cut to represent the size of the normal heart at distances from 1 to 6 ft. He found that allowance could be made for the distortion, and gave figures to illustrate the point. For distance radiography or radioscopy a special screening stand is required; one of the best is that used by Professor Forssell, of Stockholm. Incorporated into it is a movable tube box, which allows of it being used for orthodiagraphy. A large number of papers have been published giving full details and measurements of the normal and the pathological heart.

#### (5) *Instantaneous Radiography.*

This is of the greatest possible value. It shows the heart in all phases of activity when a number of plates are exposed in rapid succession. The method may be used at any distance, the most usual being at 3 ft., when a fairly accurate picture is produced, or at a distance of  $2\frac{1}{2}$  metres, when the size of the heart may be regarded as an actual drawing of the organ. When combined with orthodiagraphy a very complete record of the heart is obtained.

The following is a very interesting example of the value of instantaneous radiography, published by Speder (*Archives d'Electr. med.*, 1914, xxiv, p. 14). He presents a remarkable case of hydropericardium with pneumothorax following on traumatism. An instantaneous radiogram (one two-hundredth of a second) from a distance of 1 metre was obtained by means of the single flash apparatus of Dessauer, ten days after the accident, and in spite of the patient's dyspnoea and pulse frequency (140) the contours were given as definitely as if by crayon drawings. Not only was the pericardial shadow recognizable but also the projection of the heart within the shadow. The pleural effusion in the left costo-diaphragmatic sinus was indicated, as were also the pneumothorax and the retracted lung. The radioscopic examination and the instantaneous radiograph were repeated several times, and marked the gradual return to a normal picture, which corresponded with the clinical improvement.

The author states that the visibility of the heart in certain cases of pericardial effusion has been contested, but on several occasions with instantaneous exposures he has been able to demonstrate the visibility of the heart, when the shadow of the pericardium has been distended by reason of fluid (hydropericardium).

#### PROFESSOR GÖSTA FORSELL'S DEVICE FOR ORTHODIAGNAPHY IN CONJUNCTION WITH FLUOROSCOPY.

For the purpose of Röntgen fluoroscopy, in the standing and sitting position, and for orthodiagraphy, in conjunction with the fluoroscope, Professor Forssell has used since 1907 a modification of Holzknecht's screening stand, for which he has devised the following arrangements for the improvement of the technique.

The stand, fig. 3, is constructed in two units, the back one stationary, the front one movable. *The back unit* supports the well-protected tube-box which, with two long handles, is movable in guides, and can be fixed in every position, by means of a locking-device. The tube-box is counter-balanced by weights that slide in both the pillars supporting the tube-box. The shutter is movable from the right handle of the box. *The front, movable unit* supports both the patient and the screen. This unit is in the form of a supporting-wall with a foot-plate and is easily moved on wheels running on rails, of 3 metres long, laid in the floor.

The patient stands on the foot-plate connected with the supporting-wall, or sits on a revolving chair (fig. 3). On the front side of the supporting-wall there are arms attached to the guides, in order to bear the screen-holder. These guides are constructed so as to allow of the screen being fixed in every conceivable position. The screen is counterbalanced by weights, which slide into slots in the pillars, between which the supporting-wall is fitted in. The screen is movably adjusted within a screen-holder in the form of a metal frame enclosing a thin sheet of parchment, and can easily be removed and replaced by a cassette which is then placed against the parchment sheet by means of metal springs.

In fluoroscopy, the front unit is pushed close up to the back one (fig. 3). When radiograms are to be taken, the tube is centred during fluoroscopy,



FIG. 3.—Screening-stand, with patient in position.—(Professor Gösta Forssell.)

as desired, and the screen-holder is fixed, after which *the entire front unit with the patient and the screen* is moved quite undisturbed, as far away from the tube as may be desired (fig. 3). The movement takes place very easily. The advantage of being able, during fluoroscopy, to centre the tube and conveniently to bring the patient to a standing or sitting position at the required distance, without altering his position to the screen, will be obvious. Thereby it is also possible to ascertain by the screen whether the tube has been correctly centred and the picture properly limited.

It also embodies an indirect advantage, as such a distance can be chosen that the required picture of a thorax or an abdomen falls within a plate of 30 by 40 cm., thus larger plates being as a rule avoided. Radiographs of lungs are usually taken at a distance of 150 to 200 cm. from the anti-kathode, this depending on the breadth of the focus of the tube employed. Radiographs of the heart are taken at a distance of 200 cm.

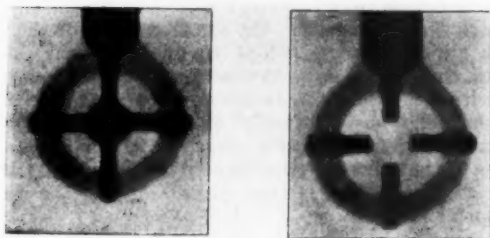
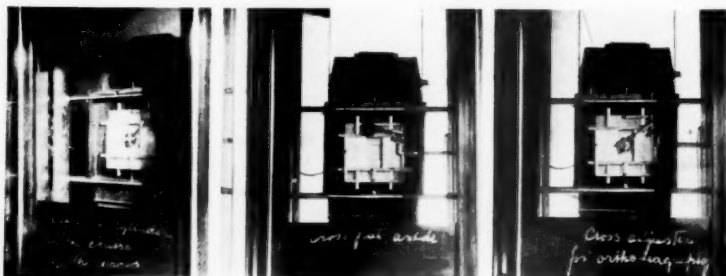


FIG. 4.—Showing construction of metallic indicator described in text.—(Forsell.)



A B C  
FIG. 5.—A, Centring cylinder in the centre of the cross; B, Cross put aside;  
C, Cross adjusted for orthodiagraphy.



FIG. 6.—To illustrate Forsell's method of orthodiagraphy.

The screening stand has the advantage over other screening stands, which are likewise provided with a movable screening unit and a tube-box unit, inasmuch as the movable, vertical supporting-wall furnishes a firm support to the patient, as well as to the screen and the plate-holder.

In the employment of orthodiagraphy in conjunction with fluoroscopy, Forssell, in agreement with Holzknacht, uses a lead cross applied to the middle of the aperture of the diaphragm. This cross is lowered in front of the diaphragm, opening by a simple gearing from the right handle of the tube-box. By fixing four leadpieces on an aluminium ring, he has made a cross with free centre (fig. 4). This arrangement has been designed because the lead cross must be made rather strong, in order to be seen through stout patients, and therefore the centre of the cross often covers just that contour that is to be orthodiographed, whereas if the cross has a free centre, one can, on the contrary, see the contour in the centre very well, whilst the arms of the cross clearly denote the location of the central rays (fig. 5).

In order to centre the cross carefully, for orthodiagraphy, opposite the focus of the Röntgen tube, Forssell has had a simple centring apparatus made (fig. 5). A brass tube of 75 mm. in length, in both ends of which a lead diaphragm of an inner diameter of 1.5 mm. is fitted in, is slipped into the free space in the middle of the cross, in which the centring-tube exactly fits.

By the centring the lead cross is lowered and the centring tube is fitted into the centre of the cross (fig. 5), after which the fluorescent screen is adjusted in frontal direction, being controlled by a ratchet in the screen-holder.

The Röntgen tube is placed in the tube-box in such a way that the focus is approximately laid in the middle of the centre of the diaphragm. By means of two screws with isolated handles, a careful adjustment of the cross can be made. The fixing is adjusted, until the aperture of the centring tube, in approved manner, leaves a circular picture on the screen. After this the centring tube is removed. If the cross is carefully adjusted, the middle of it will give the exact position of the central ray. One soon gets accustomed to perform the orthodiagraphic drawing quickly and surely.

By controlling orthodiagraphy of metal objects, Professor Forssell is convinced that a very exact determination of the surface measurement can be made by means of this orthodiagraph (fig. 6). It has the great advantage of enabling one to draw with a relatively large field of vision, and the orthodiagraphy can be quickly carried out, in conjunction with fluoroscopy, whilst the operator is as well protected as possible.

At the fluoroscopy and the orthodiagraphy the operator is protected behind a screen of 3 mm. lead covered on both sides with veneer, this screen being movable on the same rails on which the front unit is running.

Vaquez and Bordet ("Le Cœur et l'Aorte: Études de Radiologie clinique," Paris, 1913) give the result of systematic investigations at the Hôpital Saint-Antoine. They lay great stress on the necessity of using precise radiological methods, and the combined use of teleradioscopy and orthodiagraphy is held to provide more important data than the teleradiographic plate. The authors recommend that the anti-kathode be at a distance of not less than 2½ metres—only approximately parallel rays thus being used. Under these conditions an object of 15 cm. width is enlarged only from 4 to 5 mm. The authors describe the appearances of the normal heart in various positions and the changes brought about by various disorders.

At a later date these authors presented a monograph on the X-ray examination of the heart, which gives in full detail the technique described by themselves; a perusal of its pages will demonstrate very clearly to what an advanced stage cardiological technique has developed.

Woodburn Morison and Leonard M. White published in the *Archives of Radiology and Electrotherapy*, 1918-19, xxiii, p. 282, a paper on "A Radioscopic Method for estimating Hypertrophy of the Left Ventricle." They

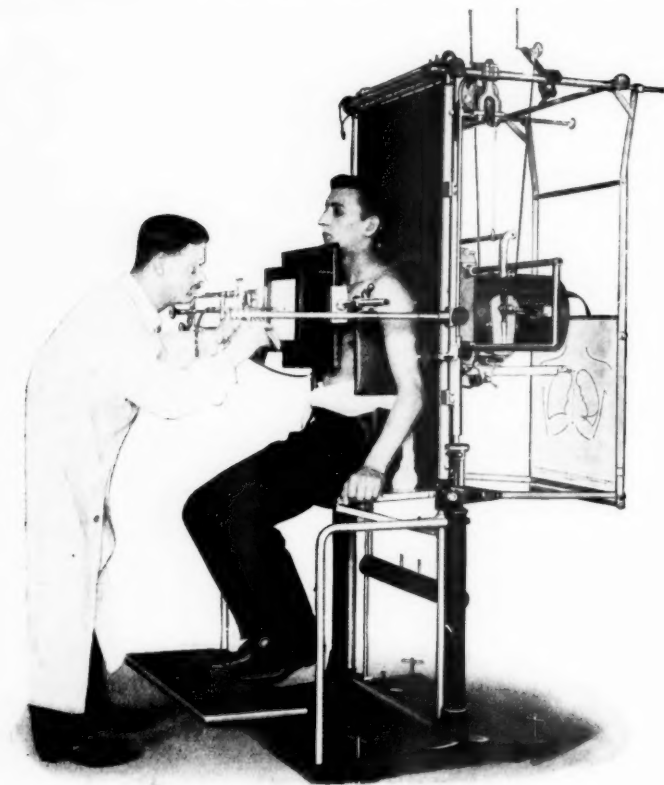


FIG. 7.—Orthodiagraphic apparatus.—(Scholl.)

begin by pointing out that the left ventricle forms a very small part of the anterior surface of the heart, and that the bulk of it lies deep, and in the mediastinum. It follows then that in order to diagnose commencing hypertrophy one must determine the increase of the ventricle in depth.

The technique is based on the method of similar triangles, first utilized by Mackenzie Davidson for locating foreign bodies, but there are certain features which require detailed consideration.

If the apex of the heart were a point, or an object of slight thickness, the depth could be easily calculated by the method above referred to, but this of course is not the case. Reference to fig. 1 (p. 19) will readily explain where the difference lies. Here the increase in size of the ventricle is deep in the mediastinum, and the apex of the heart is not altered in position. On shifting

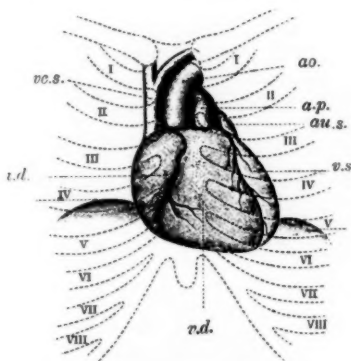


FIG. 8.—Anatomical drawing showing position of heart and great vessels to anatomical structure of thorax.—(After Assmann.)

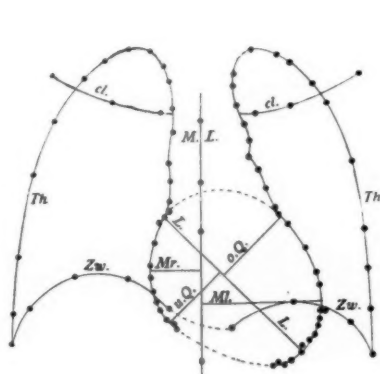


FIG. 9.

FIG. 9.—Orthodiagraphic tracing.—(Groedel.)

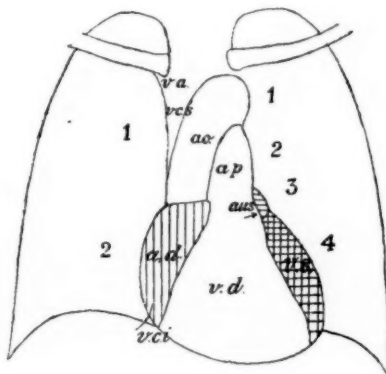


FIG. 10.

FIG. 10.—Schematic drawing to show position of chambers of heart in a skiagram. (After Assmann.)

the tube 10 cm. to the left, the rays meet the border of the heart sooner than in the case of the normal heart, and the edge of the shadow is projected outwards. The deflection is considerable, and indicates an increase in the size of the ventricle in depth. It can be understood from this that the method reveals not the depth of the apex, but the furthest projecting edge of the



ventricle in the mediastinum. By similar means it is possible to obtain an approximate idea of the size of the right ventricle, but here the difficulties due to anatomical considerations become more marked. There are, however, other radioscopic methods of determining the size of the right ventricle, viz.,

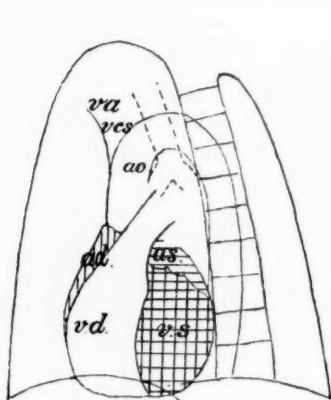


FIG. 11.

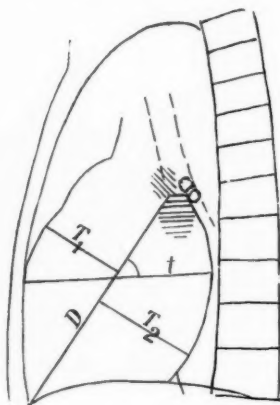


FIG. 12.

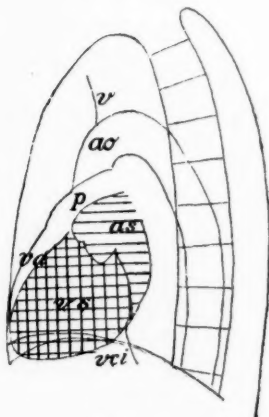


FIG. 13.

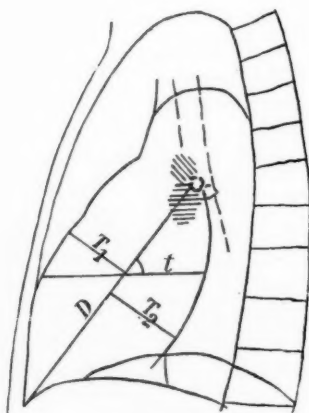


FIG. 14.

Diagrams of oblique lateral views of chest to show position of heart and great vessels in various abnormal conditions.—(After Assmann.)<sup>1</sup>

increase in size of the transverse diameter, bulging of the diaphragmatic portion of the lower edge of the right side of the heart, which gives sufficiently clear indications. The technique is simple and rapid. Either the vertical or the horizontal position can be used, but the authors have followed Dr. Vaquez

<sup>1</sup> These figures also illustrate a method for the measurement of cardiac outlines in this position.

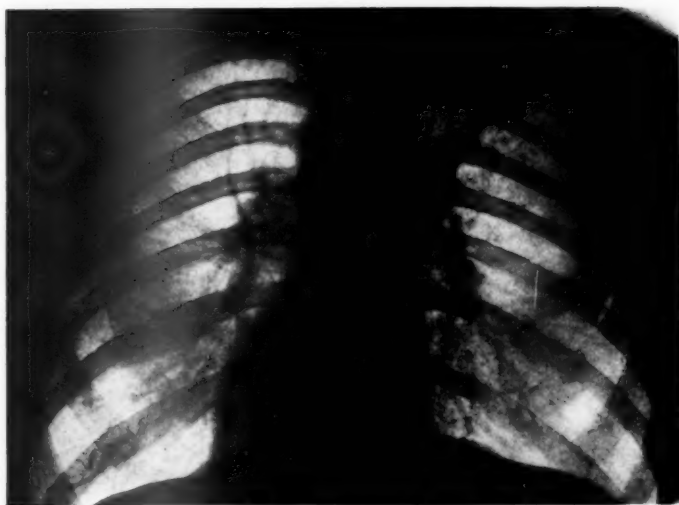


FIG. 15.—Pulmonary tuberculosis. Instantaneous radiogram—200 cm. distance between plate and anticathode.—(Forssell.)



FIG. 15A.—Miliary tuberculosis. Same conditions as in fig. 15.—(Forssell.)

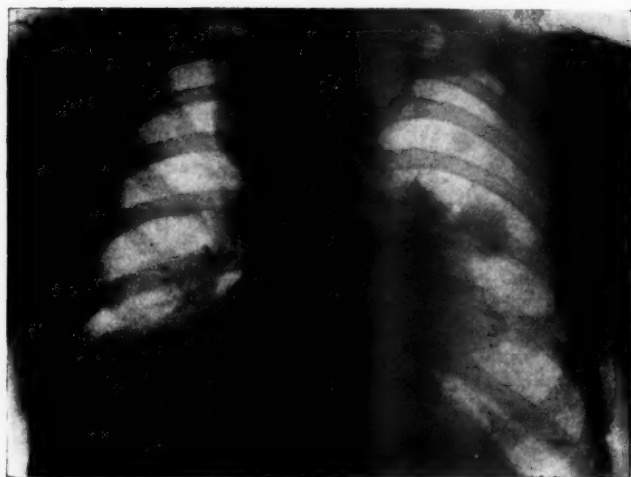


FIG. 16.—Sarcoma of the lungs and mediastinum. Distance between plate and anti-cathode, 200 cm.—(Professor Gösta Forssell.)

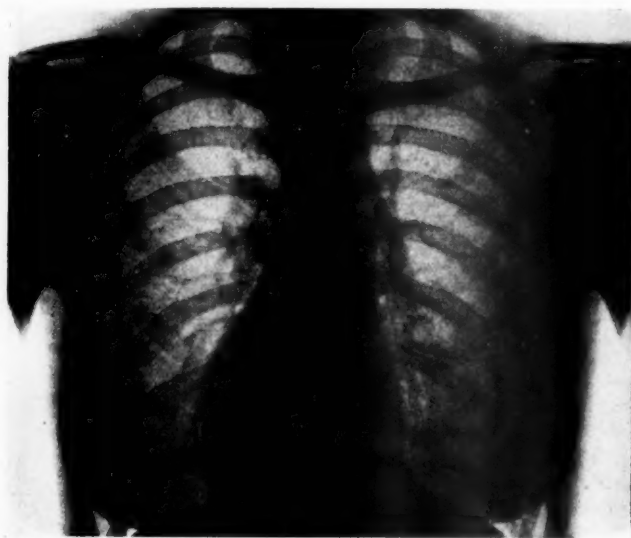


FIG. 17.—Normal thorax. Distance, 200 cm.—(Professor Gösta Forssell.)



index" might be used, qualifying it if necessary, e.g., "cardio-radiographic index, left ventricle, 12 cm."

To facilitate the procedure a simple piece of apparatus has been devised by Dr. Woodburn Morison and Dr. Leonard White which can be adapted to any screen, and which, among other advantages, does away with the necessity of using a grease pencil, and renders the operation somewhat more accurate. It consists of a wooden frame (1) which supports two sliding frames (2) (3). In frame (2) are inserted two tightly drawn parallel wires, A, B, which are exactly 10 cm. apart. At the edge of the frame (3) is a third wire, and at (4) is a millimetre scale. The apparatus is clamped to the back<sup>1</sup> of the screen, and the patient placed in approximate position. With the diaphragm wide open the position necessary for the subsequent tube shift is obtained. The diaphragm is then shut down to a narrow slit, and the edge of the apex is brought into the centre of the band of light. By manipulating the screw to the left, wire B is made to coincide with the apex shadow. The tube is then shifted to the left until the wire A appears in the centre of the beam of light. The diaphragm is then opened wide, and, by means of the screw to the right, wire C is shifted to correspond with the edge of the apex, and the index is rendered very easy and rapid (fig. 18).

A series of observations on clinically normal hearts gave a range of figures which corresponded to those obtained by Dr. Vaguez and Dr. Bordet for normal hearts, viz., 7 to 14 mm., with an average of 10 mm. The following is a table of some abnormal cases, in which the highest figure was 42 mm.:—

Disease	Systolic pressure	Index	Remarks
(1) Chronic bronchitis and emphysema	160	25	Some enlargement of right heart; no valvular lesion
(2) Chronic nephritis ... ..	200	42	—
(3) Chronic nephritis ... ..	224	40	Base of left ventricle enlarged
(4) Chronic nephritis and bronchial asthma	170	17	Some enlargement of the right ventricle; no valvular lesion
(5) Chronic nephritis ... ..	180	19	—
(6) Tachycardia with dyspnoea and poor exercise response	125	11	No valvular lesion; condition undiagnosed
(7) Chronic nephritis ... ..	182	16	—
(8) Chronic nephritis ... ..	192	20	—
(9) Chronic nephritis ... ..	—	21	Phthalein output two hours
(10) Chronic nephritis ... ..	180	40	—
(11) Bronchial asthma ... ..	?	19	—
(12) Chronic nephritis ... ..	—	23	Has had hemiplegic attack
(13) Tachycardia with syncopal attacks	130	12	No valvular lesions; condition undiagnosed
(14) Mitral regurgitation ... ..	?	17	Symptoms slight; no clinical evidence of enlargement
(15) Mitral regurgitation ... ..	?	20	Discharged from the Army V.D.H.

In addition to the above cases, in all of which, with two exceptions, there are raised indices, a number of cases of nephritis with few signs and symptoms apart from depressed renal function have been examined. These indices will be periodically taken to record their progress.

The value of this method should lie in the evidence it gives of a hypertrophied left ventricle before ordinary clinical evidence reveals such a condition.

<sup>1</sup> Dr. Morison informed me that the frame is now clamped on the front of the screen. This enables the observer to make direct readings.

As stated above, a few cases have been met with in which it has been possible to say that hypertrophy exists, but in which ordinary screening, percussion, and palpation gave no evidence of the hypertrophy; but it will be necessary to follow up such cases in order to obtain a proper prognostic value of such slightly raised indices.

#### THE X-RAY CARDIOGRAM.

The next step in the evolution of X-ray cardiological technique is an important one, which when thoroughly developed may prove to be of very great value.

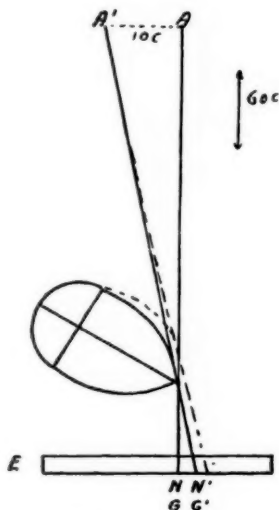


FIG. 19.—To illustrate method of Vaguez and Bordet.—(Morison and White.)

Gött and Rosenthal (*Münch. med. Woch.*, 1912, lix, p. 2033), describe röntgenkymography as an X-ray method of demonstrating the movements of the heart. The patient stands with his back to the X-ray tube, with his chest leaning against the lead screen, in the middle of which is a narrow horizontal slit at about the level of the left nipple. When the image through the slit is examined with the fluorescent screen it is found to be divided into two parts—a transparent part corresponding to the lung and a dark part corresponding to the shadow of the left ventricle. The line of junction between the light and the dark field is in continuous movement, making short excursions to the right and left, in correspondence with the movements of the ventricle. If a photographic plate be moved across the slit a series of pictures of the slit will be obtained, each of which will show a different position of the line between dark and light, thus furnishing a graph representing the movements of the ventricle. Such a skiagraphic production of the curve of movement is called a röntgenkymograph. By the use of more than one slit simultaneous movement curves

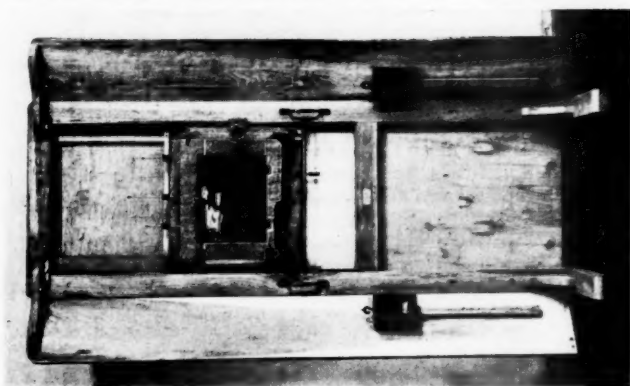


FIG. 20.

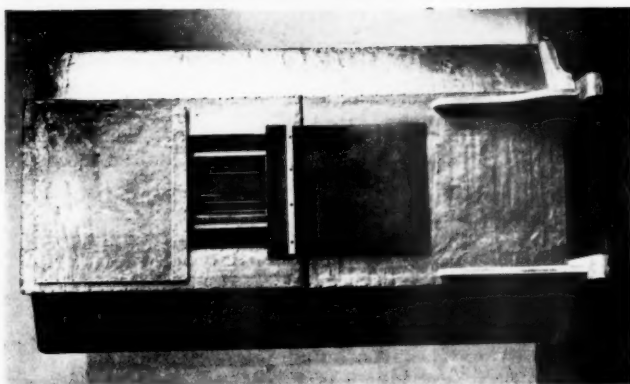


FIG. 21.

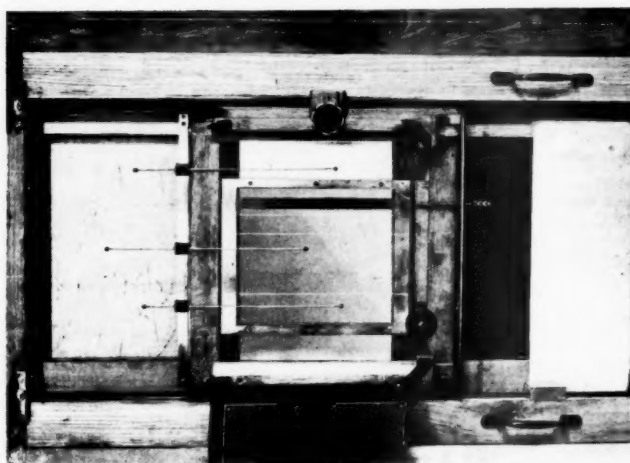


FIG. 22.

Photographs of screening unit of upright screening stand to show details of the mechanism.



of the left and right ventricles, the aorta, and the pulmonary artery may be obtained.

For taking the kymogram a Polyphos universal inductor with a rapid switch was used. This illuminated a Rosenthal iridium tube for four or five seconds without altering its hardness—a time quite sufficient to obtain four or five pulsations of the heart. The plate was 24 cm. by 30 cm., and was used with an intensifying screen, or a film with an intensifying screen on either side of it might be used instead of the plate. The plateholder was moved vertically across the slit by clockwork. The slit was 3.5 mm. across and the focus distance 50 cm.

Crane (*American Journal of Röntgenology*, 1916, p. 513) describes the röntgenocardiogram, which is a tracing of the heart's beat obtained by covering the heart with strips of sheet lead so arranged that a narrow slit, usually about 2 mm. in the lead can be adjusted over the pulsating margin of the cardiac shadow as seen in the screen. Several of these slits can be adjusted simultaneously over different areas of the pulsating heart, the aorta, and the pulmonary vein. A photographic film on a screen in a cassette is then made to travel at a given speed, usually about 5 cm. per second, transversely over these slits, while the Röntgen ray is turned on. The result is a tracing which is similar to and comparable with a sphygmogram, a polygram, or an electrocardiogram. It deals directly with the heart muscle, and is capable of giving a separate record of the muscle movements of each chamber of the heart, in which respect it stands unrivalled.

The essential points in X-ray examination are: (1) The detection of dilatation or hypertrophy of certain chambers of the heart; (2) the character of the pulsation of various heart chambers; and (3) synchronism of different chambers.

The disadvantages of the röntgenoradiograph are: (1) Heavy patients often offer resistance to the passage of the rays; this is overcome by using wider slits, a slower movement of the carriage, and a ray of higher penetration; (2) some patients with cardiac disease cannot lie in the horizontal position; this can be overcome by the sitting position, in which the patient is placed with the back to the lead screen of the cardiograph, the tube being in front, and each slit adjusted over an interspace between the ribs; (3) patients with advanced tuberculosis, &c., may not be suitable for Röntgen tracings because the cardiac outline is obscured or actually concealed.

The author describes the X-ray appearances seen in each of the valvular diseases of the heart, and also various less frequent cardiac conditions, which may or may not present definite radiographic appearances, such as auricular flutter, heartblock, &c.

A consideration of the paper by the later writers led one to the conclusion that if a complete scheme could be introduced it would go a long way towards standardizing cardiac technique, and possibly popularize the method by demonstrating its great utility. It occurred to me that if the moving plate method could be combined with a time record it would furnish a valuable confirmatory record in favour of the electrocardiographic method by demonstrating radiographically what the electrocardiograph shows by an electrical method. Accordingly, in order to increase the value of the method described by the French writers, an apparatus has been constructed which, while combining the measurement of the size of the left ventricle, allows of standard positions for the heart, and allows also of the ready location of the heart outline in relation to fixed anatomical landmarks (figs. 20-23A).

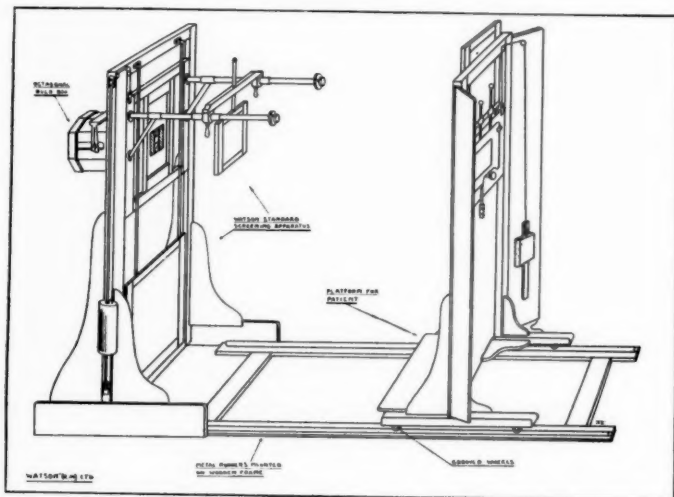


FIG. 23.—Upright screening-stand in two units.

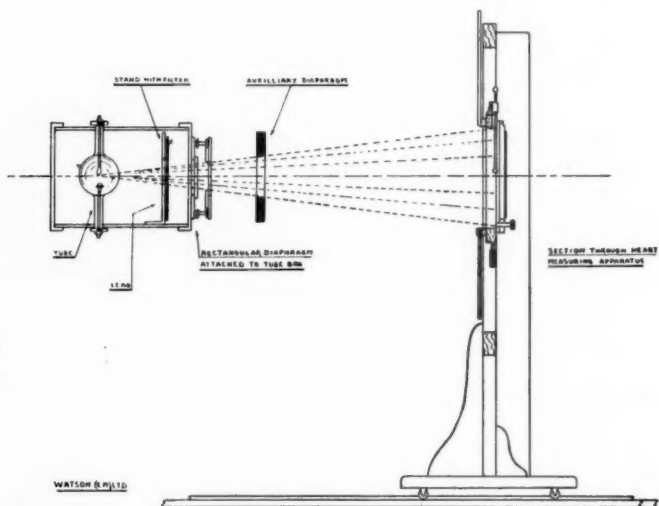


FIG. 23A.—Diagram to show arrangement of tube, diaphragm and screening unit. This apparatus can be used for distance radiography and stereoscopic work.

The addition to this apparatus of an attachment to the tube-box of a mechanism similar to that introduced by Professor Forssell would allow of orthodiagraphic tracings being made, and would greatly add to the completeness of the apparatus. It therefore follows that the screening-stand adapted for a complete examination and recording of the condition of the heart must be rather complex, for it must be capable of allowing of an examination by one or all of the following methods: (1) Radioscopy; (2) stereoscopic radiography; (3) radioscopy combined with actual measurements of the heart borders by (a) orthodiagraphy, (b) Vaquez and Bordet's method; (4) radioscopy with radiography by use of the moving plate and slit diaphragm; (5) recorded movements of the heart by means of fixed anatomical points and timing apparatus.

To complete the record a standard chart has been constructed which, when placed over a tracing made upon the fluorescent screen, will give a visual demonstration of the outline of the heart, and show at once in what respect it differs from the normal.

To make a standard chart for use in measuring the heart shadow it was thought that if a normal thorax were taken and a drawing made from it of the heart shadow, it would be possible to compare it quickly with a case under observation. The outline drawing along with the principal anatomical landmarks was filled in with pliable wire, and a chart obtained, which could be placed in front of the patient, and a shadow of the wire outline obtained on the fluorescent screen.<sup>1</sup> The practical application, however, was very difficult, and it was quickly abandoned. It was very difficult to adapt the model to the average chest with any degree of accuracy.

The next step in the development of the method was to outline the heart shadow on the fluorescent screen, make the necessary measurements and then superimpose on the tracing a standard drawing on celluloid or tracing paper. Definite landmarks on the tracing were placed upon corresponding marks pencilled in from the tracing of the patient, and the two compared. To facilitate rapid measurement and comparison of differences the standard tracing was squared out in centimetres. For demonstration purposes the tracing from the patient can be made upon a paper which has all the squares recorded upon it. It is now easy to furnish the clinician with a very accurate record of the screen appearances of the patient's heart and blood-vessels.<sup>2</sup>

When a radiographic record is required, all that it is necessary to do is to place in front of the patient's anterior chest-wall a wire frame squared in centimetres. This will give a record on the plate, and deductions can be quickly made from the radiograms. It might be more convenient to place the frame in the cassette close to the plate or film.

From what has been said it is obvious that if we could work at a distance of  $2\frac{1}{2}$  metres the tracings or radiograms obtained would be free from distortion; accordingly the apparatus is so constructed that it may be used at distances varying from  $2\frac{1}{2}$  to 8 ft. between the anti-kathode and the screen or plate; the movements can be arranged for by placing the recording apparatus upon rails as is done in the screening stand of Wenkebach and Forssell. The screening stand in general use could be used in conjunction with the recording stand if it had a stereoscopic movement of the tube-box. The protected front of the

<sup>1</sup> Woodburn Morison and White. *Archives of Radiology and Electrotherapy*, 1918-19, xxiii, p. 282.

<sup>2</sup> I am indebted to Dr. Wainwright for several valuable suggestions in this part of the work.

tube-box should have a very small aperture, just sufficient to allow the beam of X-rays to illuminate the screen to a sufficient size for outlining the heart. This is of some importance from the point of view of the protection of the patient and operator. Inside the tube-box a lead screen is placed of 3 or 4 mm. thickness of lead with a small aperture in front of the tube; a filter of 3 mm. of aluminium covers the aperture. (This is shown in the diagram, see fig. 23A.) The metal diaphragm in the tube-box is essential, for it is necessary in our investigations to close down the vertical parts to a narrow slit. When working at 6 or 8 ft. distance it is well to place midway a large lead screen with an aperture sufficiently large to allow of the passage of the beam proceeding from the X-ray tube. In this way any peripheral rays not required may be obstructed. A seat consisting of a piano stool or a bicycle seat should be provided, and either of these should be easily raised or lowered to accommodate the patient.

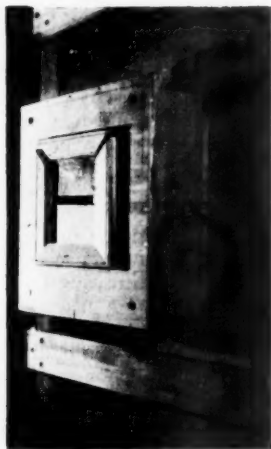


FIG. 24.—Lead "slit" diaphragm in front of the tube, and immediately behind the patient a narrow slit serves two purposes: (1) It ensures sharpness in the tracing. (2) It protects or rather limits the area of the patient exposed to the rays—a very important point in this class of work.

#### THE RECORDING APPARATUS.

This is placed in position in front of the patient. It consists of a frame which is movable in the horizontal axis, along an outer supporting frame; a handle with a rack action controls the movement of the inner frame in either direction from right to left or vice versa. The supporting frame with its attachments is suspended from the top of the upright frame, and is readily moved up and down by hand. It is counter-weighted to facilitate this movement, and can be clamped in the proper position by a hand-screw placed on the right side. A little above the moving frame is placed a horizontal metal bar having upon it three sliding metal blocks. Each of these has a small screw and a rounded terminal. Passing vertically through each block is a fine metal

rod with a small ball at either end. Inside the horizontal moving frame are several wires arranged in a manner similar to that described by Dr. Woodburn Morison and Dr. Leonard White. The wire on the right hand of the observer is attached to a fine rack, which allows of rapid movement of the wire in either direction from right to left and vice versa. The three vertical wires with metal buttons are used to indicate anatomical landmarks, and may be placed over any pre-determined point. In front of the frame a fluorescent screen is attached; this has a lead glass front, and is hinged to the left side of the apparatus (fig 24). A cassette for the plate or film is fitted to the front of the

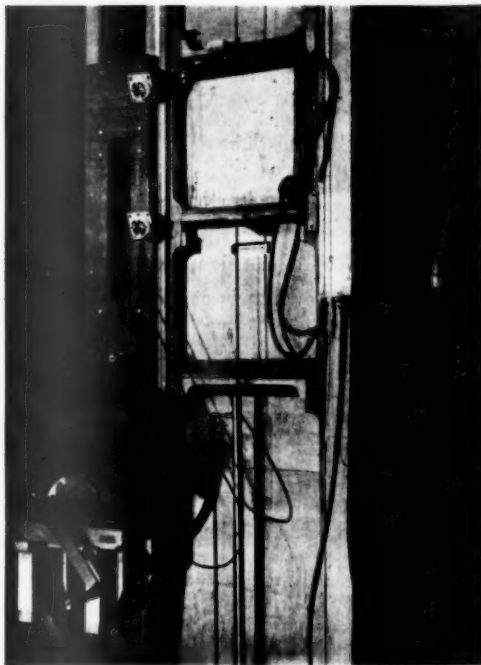


FIG. 25.—Photograph of mechanism for moving plate work. Showing cassette in position above the slit in the lead diaphragm.

frame and is easily removed when not required (fig. 25). The apparatus is therefore ready for fluorescent screen examination or radiographic records. When employed for fluorescent screen examination a grease pencil can be used for marking the outline, degree of development, &c., and these marks can be transferred to tracing paper or celluloid at leisure.

To facilitate rapid output scaled paper has been prepared: this can be fixed on the front of the screen by a small metal frame and the tracing made directly upon the paper. The paper has upon its surface a number of squares,

each 1 cm. or inch. This arrangement is useful for rapid measurements. If it is necessary to get a record of the squares upon a plate, a metal frame made up of squares can be used on the screen and plate.

The records required skill and experience in their interpretation, but as time progressed the value of the new aid became established, and new technique was introduced to facilitate the examination.

The apparatus described should furnish us with information regarding the size of the heart, the degree of hypertrophy existing, and a record of the movements of the edge of the organ. The record of the movements of the edge of the heart in derangements of the heart's action should furnish useful evidence. The scaled chart provides a record of the actual size of the organ, and should facilitate measurements, while the possession of a record of this kind will be of value when examinations are made at a later date, by providing evidence of improvement or the contrary.

The record of the actual movements of the walls of the chambers of the heart should be valuable for comparison of the record made by the electrocardiograph; together they will furnish valuable evidence for the consideration of the clinician. For making observations on the moving edge of the heart the apparatus may be modified by placing in the moving frame a series of lead diaphragms which by wire attachments may produce the slits described by Gotch, Rosenthal, and Crane.

#### THE MOVING FILM MECHANISM.<sup>1</sup>

The movement of the plate or film is obtained by an accessory piece of apparatus, which allows of the plateholder coming into close contact with the slit diaphragm.

The plate or film with intensifying screens is placed in a holder which has attached to it a mechanism for moving the plate at the required rate. The movement of the holder must be very uniform, and it should be possible to vary the speed at which it travels to suit the case under consideration. The plate-moving mechanism for use with the electrocardiograph is admirably suited to the purpose, and this is the mechanism it is intended to adopt. In addition it is intended to have a timing mechanism attached in a suitable place to allow of a time record being obtained while the exposure is made.

The radioscope and radiographic methods appear to give fairly precise information regarding the movements of the heart, changes in position and in size. These in conjunction with the sphygmogram, the cardiogram, and blood-pressure observations are of great value to the clinician.

The record of the progress of the technique for the investigation of the heart by physical methods is inspiring. It illustrates how thoroughly the earlier workers grappled with problems which to them must have seemed at times insurmountable. We owe a great debt to these pioneer workers. It is our duty to continue their work and to develop our technique to its fullest extent.

The late Professor Wertheim Salamonson was employed up to the time of his death in the arranging of apparatus by means of which he could transmit the sounds of heart murmurs, and render them audible by the use of wireless telegraphy and a phonograph. It is to be hoped that the work he just failed to accomplish has been left in such a state of advancement that some other

<sup>1</sup> I am indebted to Mr. P. G. Neate for valuable suggestions and practical help in the design and construction of this part of the apparatus.

worker will be enabled to continue it. This achievement, if it becomes practicable, may lead to further developments in murmur analysis, and when it is possible to obtain a faithful reproduction of the heart movements on a moving film a further step will have been made, for then the clinician may study at his leisure the complicated movements of the active heart.

The great clinicians of the past paved the way for these later developments. Surely no example exists which so thoroughly proves the value of continuous effort along definite lines.

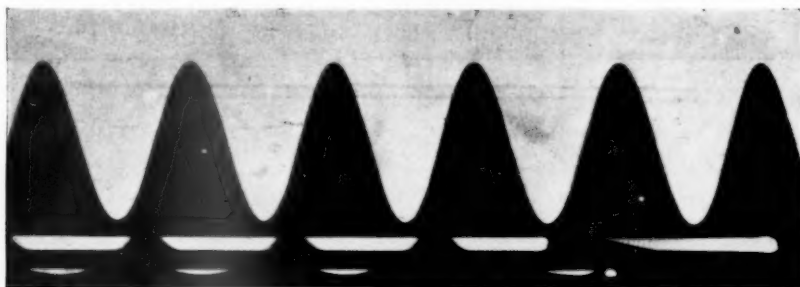


FIG. 26.—Swinging pendulum: Period, 10 secs.<sup>1</sup>

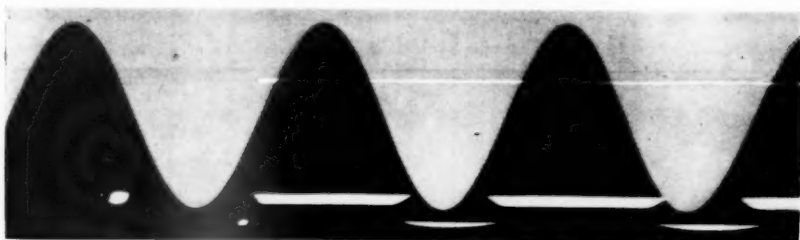


FIG. 27.—Swinging pendulum: Period of moving plate about 4 secs. Film showing periodicity of transformer machine at slower speed. Record of timing arrangement shown in lower part of figure.



FIG. 28.—Swinging pendulum, 70 per minute. Showing the variable skiagraphic appearances of altered periodicity of falling plate.

<sup>1</sup> Figs. 26, 27, 28: Records of experiments conducted with a slit diaphragm and swing pendulum.



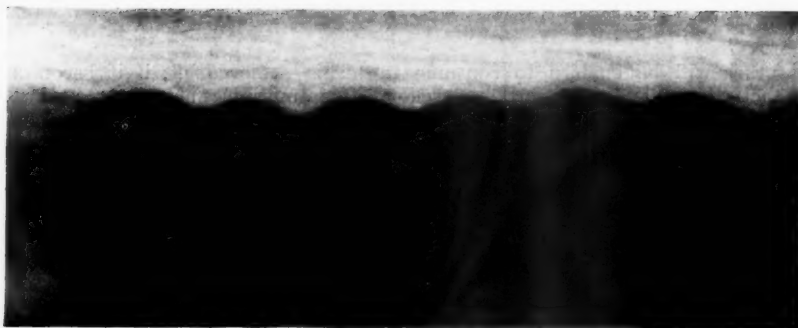


FIG. 29.—Ventricular beat. Period of moving plate, 7 secs.<sup>1</sup>

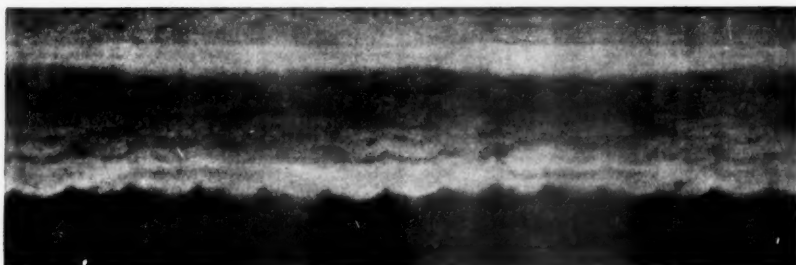


FIG. 30.—Ventricular beat, showing a periodic irregularity. Period of moving plate, 12 secs.



FIG. 31.—Ventricular beat. Period of moving plate, about 8 secs.

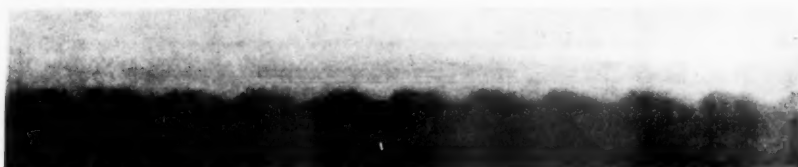


FIG. 32.—Ventricular beat. Period of moving plate, about 8 secs.

<sup>1</sup> Figs. 29-38 are records obtained by the slit diaphragm method showing the moving edges of the heart in a number of normal and abnormal conditions.

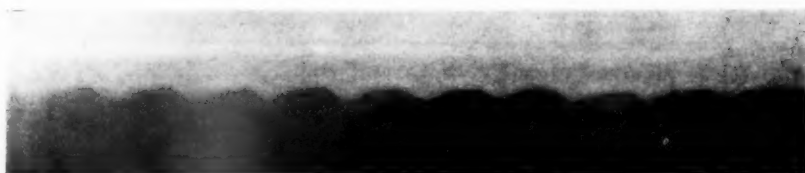


FIG. 33.—Ventricular beat. Period of moving plate, about 8 secs.



FIG. 34.—Ventricular beat, showing cardiac irregularity. Period of moving plate, 10 secs.

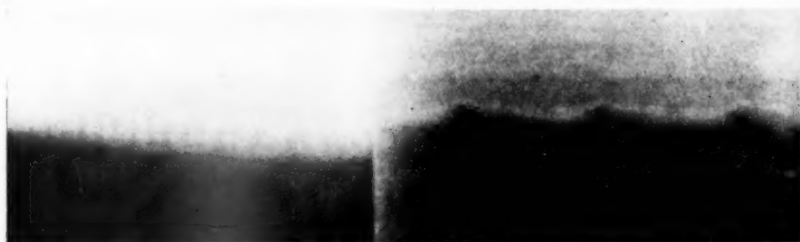


FIG. 35.—Ventricular beat. Plate moving slowly, 50 secs. ; then more rapidly 7 in. in 7 secs.

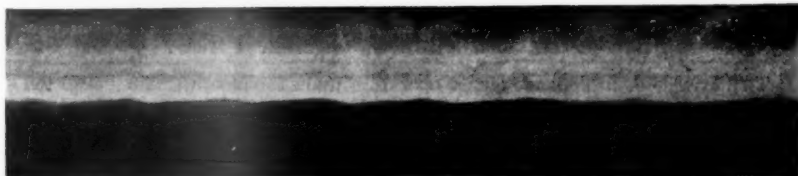


FIG. 36.—Showing movement of right auricle.

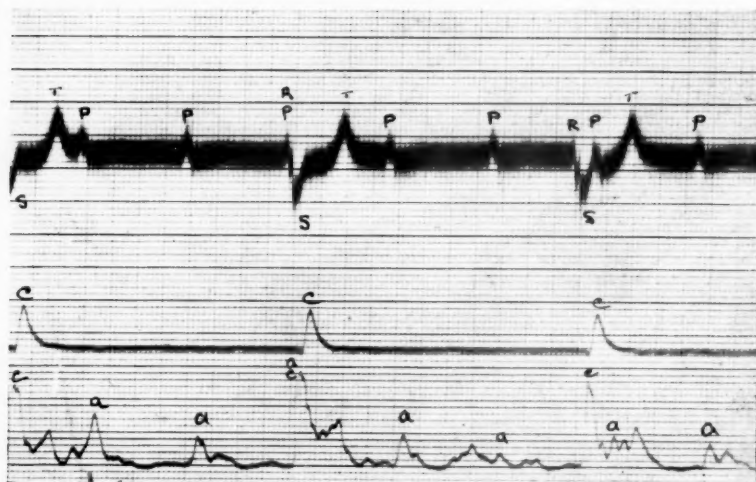


FIG. 37.—Electrocardiogram from same case.—(Dr. Wiltshire.)

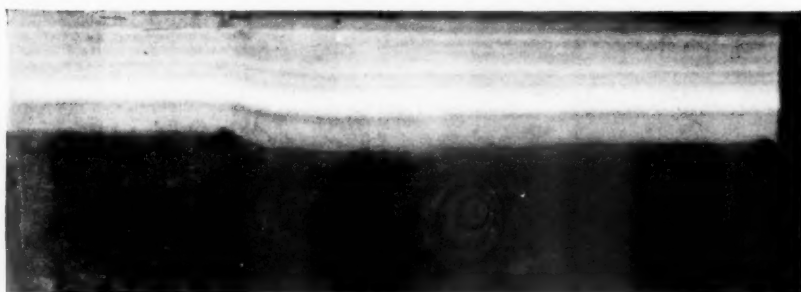


FIG. 38.—Ventricular beat, showing heart block (Stokes-Adams disease); 28 per minute. Period of moving plate, about 4 secs.

### Apparatus for Recording X-ray Doses.

By G. E. S. PHILLIPS, O.B.E., F.R.S.

THE arrangement depends for its operation upon the electrostatic attraction of a movable metallic disc, which is maintained at a constant potential, by a stationary brass plate the potential of which is varied by connexion to an ionization chamber held in the path of the rays. The apparatus, operated by the ionization produced in air by only 1 mg. of radium bromide is shown at work giving a sound signal (by the periodic ringing of an electric bell situated at some distance away) while simultaneously making a permanent record by actuating a series of dials.

[October 20, 1922.]

## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

### Pulmonary Tuberculosis as shown by X-rays, but without Physical Signs.

By STANLEY MELVILLE, M.D.

ONE of the greatest clinicians and pathologists of our day has lately written a book on "Pulmonary Tuberculosis." He refers to a series of combined examinations made at a military hospital—on the one hand by himself, and on the other hand by a radiologist of gracious memory—with the object, as he avers, of proving for his own work the value of X-ray examination in the diagnosis of pulmonary tuberculosis. "In no case," says the writer, "did X-rays indicate the presence of unsuspected disease." Now, such a statement, coming from one of the writer's eminence, was so diametrically opposed to my own personal experience (and I venture to say to that of most of my colleagues) that I scrutinized the sentence carefully. The word "unsuspected" seemed to me to be a word capable of many readings, and I began to wonder whether the writer might not be having a little joke at our expense. Surely, to the clinician experienced in chest work, there is much suspicion of disease even in cases which show no physical signs, such suspicion amounting almost to a certainty. If this was the meaning of the writer, then I think we shall all be in agreement with him; if his meaning was otherwise, then I think our time will not be wasted if we consider a few typical cases. I make my contribution in no spirit of criticism, but as a plain statement of fact and rather in a spirit of admiration for the honesty of great clinicians who can admit fallibility. I believe that our art is but one factor in diagnosis (though I do not minimize its importance), and that it will only be by constant "team-work" that we shall arrive at the truth.

Now, the cases which I show you to-night, without exception, are cases in which, after careful examination by competent physicians, no physical signs were found. That suspicion of tuberculous disease was present in the minds of those physicians was obvious—indeed, in some of the cases tubercle bacilli were actually present.

I do not feel competent to offer any explanation of the apparent absence of physical signs, except upon two points:—

(1) That the presence of slight emphysema tends to mask physical signs, and emphysema was present in certain of my cases.

(2) That abnormalities in the percussion note and in the breath sounds are, I believe, not readily detectable at a greater depth than about 4 mm. from the lung surface where the area of infiltrated lung is small in extent.

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The following are notes of the cases which I have selected for consideration:—

*Case I.*—Male, aged 54. T.B. negative six times. History: Cough, sputum, dyspnoea, bronchitis and pleurisy, hæmoptysis (traces only). X-ray: Both apices opaque and shrunken, heart narrow, drawn to the left. Right lung: Extensive infiltration. Left lung: Infiltration and fibrosis. Clinical (three weeks later): Final diagnosis, chronic pulmonary tuberculosis. Discharged improved. Sent to sanatorium.

*Case II.*—Male. T.B. negative. History: Malaria and dysentery while in Army.



FIG. 1.—Case II.

Demobilized; later hæmoptysis (excessive) on two occasions. X-ray: Mediastinum drawn slightly to the right. Right chest: Apex of lung opaque and mottled. Infiltration and fibrosis of right lung. Left chest: Compensatory emphysema of lung; much linear striation, and some suspicious scattered mottling. Clinical (two weeks later): Fine crepitations under right clavicle. T.B. positive. Complement fixation test positive.

*Case III.*—Male, aged 40. T.B. negative. History: 1900-1905, in Army; 1905, malaria, occasional slight cough; 1909, debilitated; 1918, onset of cough; 1919, cough worse, suspicious larynx; never pneumonia, pleurisy, influenza or bronchitis. X-ray: Lessened translucency of both apices (right more than left); general infiltration of both lungs; heart narrow and vertical. Clinical (three weeks later): A few T.B. found.

Complement fixation test negative. Slight pyrexia. Later: Diffuse infiltration of both lungs.

*Case IV.*—Female, aged 38. T.B. positive. History: No history of pneumonia or pleurisy. 1919: Influenza, cough, anorexia. No hæmoptysis. Pain in left base. X-ray: Fixation of left side of diaphragm. Heart drawn slightly to the left. Infiltration of both lungs. (?) Excavation of the left lower lobe. Clinical (later): Chronic pulmonary tuberculosis (? primary basal). Excavation left lower lobe.

*Case V.*—Female, aged 27, single. T.B. negative four times. History: Rheumatic fever. 1920: Influenza; winter cough for five or six years. 1921: Hæmoptysis.

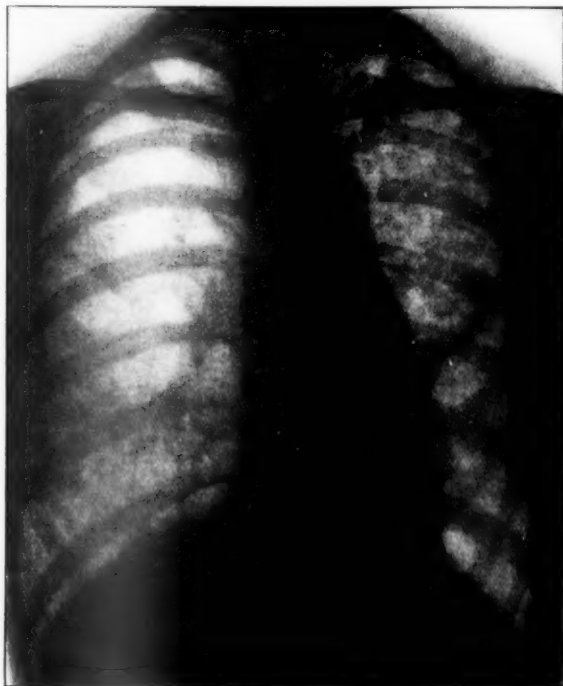


FIG. 2.—Case V.

X-ray: Right lung: emphysema; infiltration; fibrosis, chiefly middle and lower lobes. Left lung: Apical infiltration. Heart narrow and vertical. Clinical (one month later): A few crepitations at left apex. Later: Crepitations at both apices. Complement fixation test positive.

*Case VI.*—Male, aged 45. T.B. positive. History: Measles and pertussis. Cough fifteen months; lassitude and loss of weight. Prisoner of war in Germany. Suffered from septic arm in consequence of a blow on the arm by a German. X-ray: Heart narrow and vertical. Apices of both lungs opaque. Extensive infiltration of both lungs, and evidence of early excavation of right upper lobe. Clinical: Pulmonary tuberculosis both apices.

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*Case VII.*—Male, aged 20, single. T.B. negative. History: 1919-21, in Army. 1919: Onset of cough. 1920: Cough returned with bronchitis. 1921: Anorexia and loss of weight with some evening pyrexia. T.B. found. X-ray: Heart narrow and vertical. Expansion and translucency lessened at both apices. Infiltration and fibrosis of both upper lobes. Clinical (one month later): Diminished air entry at right apex. Later: Crepitations at both apices. Apyrexial. Final diagnosis: Chronic pulmonary tuberculosis (right more than left).

*Case VIII.*—Female, aged 23, single. T.B. negative. History: Cough, three weeks. No expectoration; temperature, 99'4°; weight, 7 st. 5 lb. Family history:

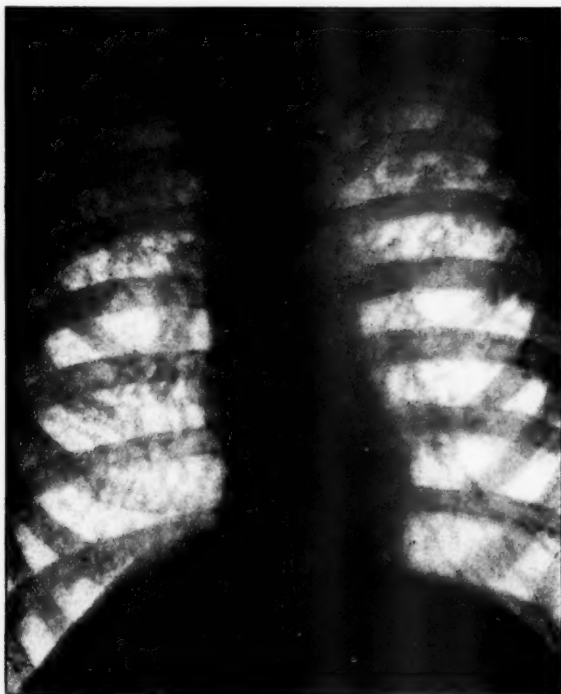


FIG. 3.—Case VI.

one brother alive, pulmonary tuberculosis. One sister died, pulmonary tuberculosis. X-ray: Heart drawn to the left, and left border ill-defined. Left side of diaphragm raised and movement restricted. Dense, irregular opacity at left base suggesting fibrosis and excavation. Clinical (some weeks later): T.B. positive. Chronic pulmonary tuberculosis, right and left (excavation left base). Died two months later.

*Case IX.*—Male, aged 38. T.B. negative. History: Pain under ribs. Morning cough: Expectoration plus, hæmoptysis streaks, loss of weight. Family history: Nothing known. X-ray: Emphysema and some shrinkage of apices. Heavy hilar shadows. Central infiltration of both lungs. Right lung: Interlobar consolidation. Clinical (final diagnosis): Chronic pulmonary tuberculosis both lungs. T.B. positive. Death.



I think one of the primary objects of our discussions here is to talk over our difficulties. Our position in the medical world and with our medical and surgical colleagues is a very different thing to what it was when many of us present began the uphill work of radiography, thanks chiefly to the strenuous work of many whose names are now household names among us. But we are not near our goal, and from time to time there are whisperings that we do not give all the assistance of which we are capable and which is expected of us; that the interest of the patient may better be served by the consultant becoming his own radiologist. I do not wish to labour the point except to say this much, that I think the consulting physician or surgeon who goes outside his province in this way, loses a subtle something of respect, fails in that fineness of outlook and judicial function, with which, possibly in an old-fashioned way, one has always invested him. Those of us who are conversant with the work of the law courts know well the analogy of the judge who is also the advocate.

With regard, however, to the question of the diagnosis of pulmonary disease, I think it is up to us to consider the position. Interpretation of the shadows in a skiagram of the lung is not so easy as is generally supposed, and I venture to say that all of us are tempted to read more into a skiagram than is justifiable. For instance, we are inclined to confuse peri-bronchial fibrosis with a true fibrosis affecting the lung tissue. It is true that in this respect we are at somewhat of a disadvantage, for usually we are asked to commit our opinion to writing after a single examination. I emphasize this difficulty, but it does not get rid of the feeling of doubt, of which we are all conscious, that the clinician may feel that we are not assisting in diagnosis as we ought. If anyone doubts the accuracy of such a statement, I would refer him to the one skiagram reproduced in Sir James Kingston Fowler's book on "Pulmonary Tuberculosis." In this reproduction not one of the classical X-ray evidences is present, and yet upon this case one of us has written a positive diagnosis of chronic pulmonary tuberculosis, and has kindly but definitely been given a rap over the knuckles by the writer of the book in question.

I have referred to the possibility of the clinician becoming his own radiologist. My hope is that the future may see the radiologist so highly trained and the clinician so keen on team work as to make so retrograde a step not only unnecessary but impossible.

## A Method for the Opaque Meal Examination of the Stomach.

By S. GILBERT SCOTT.

(ABSTRACT.)

THE value of the opaque meal examination in diagnosing pathological conditions of the stomach and small intestine is now fully recognized. There are various methods in use, the examination being by no means standardized. Until recently, radioscopy was little employed in America, diagnosis depending on evidence obtained from a number of plates, and it is interesting to note how American radiologists are now beginning to realize the value of radioscopy. On the Continent, a combined plate and screen examination is the most popular.

In England, screening is considered of value, but evidence from radiographs is still thought of greater importance. The history of the case, together with the unreliable gastric complex, is, in my opinion, given too much diagnostic

\*

[December 15, 1922.]

value. I have tested a number of methods. Practically all will indicate the presence of the grosser lesions, but are unreliable in diagnosis of very small gastric or duodenal ulcers.

Owing to the number of opaque meal examinations at the London Hospital, one's method must be rapid and accurate, and expense reduced to a minimum. Radioscopy, therefore, was the line indicated, so for several years I have worked on this definite line of technique, and as it has consistently given over 94 per cent. of correct diagnoses, I think it worthy of description.

#### SYSTEMATIC RADIOSCOPIC PALPATION.

This must not be confused with the stomach-prodding or palpation used by many workers. It consists of a minute and systematic examination of the entire wall of the stomach and duodenum. The barium cream, given as the opaque meal, is, as it were, smeared over the mucous membrane of the stomach, so that the folds are easily visualized and any defect or distortion detected. By this method, both the anterior and posterior surfaces of the stomach can be examined minutely. This is important, as with other methods, ulcers in these positions are frequently missed. This systematic palpation is carried out inch by inch, starting at the cardiac and ending at the duodenal end. The method is simple, does not involve the use of expensive apparatus or of plates, but in a busy hospital is a strain on the radiologist.

In radioscopic palpation, diagnosis depends solely on *direct* evidence and a positive diagnosis is only given when the lesion has been visualized on the fluorescent screen. The radiologist should not depend upon *indirect* evidence. Experience has forced me to rely on screen work rather than on radiographs. Certain lesions, visible on the screen owing to their position, are almost impossible to demonstrate on radiographs.

#### CLINICAL HISTORY.

Generally speaking, it is not necessary for the radiologist to know the clinical history of a gastric case. I think the diagnosis is more likely to be correct if given independently. The clinical history of a case is often unreliable and misleading. The radiologist should undertake his investigations with an open mind. Carman, of the Mayo Clinic, supports this contention: "However helpful the clinical facts may have been in the developmental stage of gastric radiology, and however necessary this still may be for the interpretation of other radiological findings, their employment at this time by the radiologist in diagnosis of duodenal or gastric ulcers is unnecessary, and would increase, rather than diminish his mistakes."<sup>1</sup>

#### DIAGNOSTIC VALUE OF RADIOGRAPHS.

I am at variance with many of my colleagues as to the value of radiographs in *diagnosis* of gastric lesions. Practical experience has taught me that radiographs must be of secondary importance; in many conditions it is possible to give a definite diagnosis from the screen examination, yet difficult, or impossible, to demonstrate the condition on a radiograph. I contend that they are unreliable in the case of small ulcers, although they will demonstrate grosser lesions. Radiographs are useful for recording unusual conditions and for demonstration of the lesion to the surgeon. A lesion demonstrated on a

<sup>1</sup> *Journ. Radiology*, iii, December, 1922.

radiograph can easily be diagnosed by radiosopic palpation, but a lesion demonstrated by palpation would not necessarily be diagnosed from a radiograph.

#### A METHOD FOR RECORDING STATISTICS.

Operative findings should be compared systematically with radiological findings. Radiologists cannot always attend abdominal operations. At the London Hospital, therefore, special forms are affixed to the report sheets; these are filled in at the time of operation by the surgeon, with particulars of the condition found. Forms are returned immediately to the department and compared with the radiological findings. The accuracy of the radiological diagnosis can thus be checked regularly.

#### RAPIDITY OF METHOD.

Radioscopic palpation is rapid and accurate in detection of small lesions. In a straightforward duodenal ulcer, for instance, a diagnosis can be given within five minutes. I have been able to complete the examination of twelve gastric cases in an hour. The radiologist should have good sight, strong wrists and forearms, suitable arrangement of screen and patient, so that palpation can be comfortably and efficiently carried out.

The accuracy of a method yielding 94 per cent. of correct findings, under hospital conditions, cannot be doubted. These statistics are based on cases which have passed through the department within the last three years, diagnosis being confirmed by operative findings or post-mortem examination. They include neither cases which have yielded to treatment nor those considered inoperable. The actual figures for 1921 are given in the *Lancet*, November 18, 1922, p. 1060. Errors in diagnosis are valuable if traced to their source. Every mistake should be carefully investigated. As success in this method is to a great extent dependent on attention to small details, I propose to mention these rather fully.

#### TECHNIQUE.

A vegetable purgative, preferably castor oil, is administered thirty-six or forty-eight hours before examination, the patient then being put on a light diet, but *not starved*. The last meal, consisting of fluids, should not be taken less than an hour before examination. The object of this preparation is (1) to have the intestinal tract as empty as possible to facilitate examination, while avoiding artificial conditions (the effect of the aperient must have passed off); (2) the patient must not feel hungry, this condition having a definite reflex action on the behaviour of the stomach. The patient should wear a special garment of washable material, free of opaque bodies, easily slipped on or off.

#### COMPOSITION OF THE MEAL.

Barium sulphate is used by most radiologists, but it is difficult to administer in a palatable form, especially to patients already suffering from gastric disturbances. I have, however, found a palatable form, which I have used successfully for some years. This barium cream, commercially known as *ramul*, is made in three thicknesses: No. 1, thick flavoured, for œsophageal cases; No. 2, medium thickness, flavoured, for gastric cases; No. 3, thin unflavoured, for opaque enemata. It is sterilized, will keep for weeks, and is ready for immediate use. The barium itself is well held in suspension. The prescription is as follows:—

No. 1, *Thick Flavoured*.—Barium sulphate, 10 oz.; saccharin, 2 gr.; vanillin, 5 gr.; gum tragacanth, 100 gr.; distilled water, to 20 oz.

No. 2, *Thin Flavoured*.—Barium sulphate, 10 oz.; saccharin, 2 gr.; vanillin, 5 gr.; gum tragacanth, 60 gr.; distilled water, to 20 oz.

No. 3, *Thin Unflavoured*.—Barium sulphate, 10 oz.; gum tragacanth, 60 gr.; distilled water, to 20 oz.

Gum tragacanth and barium sulphate should be mixed as powder, and the water added gradually. Being sterilized, the mixture can be used for other purposes, such as injecting into the sinuses or empyemas, using whichever thickness is suitable to the case.

This barium cream throws a very dense and homogeneous shadow, as it creeps into nooks and crevices, such as ulcer craters, appendices, &c.

*Quantity*.—In gastric, as in œsophageal cases, this depends on the method employed and on what the radiologist wishes to demonstrate. When the stomach is full of the meal, radiosopic palpation cannot be efficiently carried out; contents are then difficult to displace so that each section of the mucous membrane may be examined in detail, and a small ulcer, situated on the anterior or posterior surface of the stomach wall may be masked by the dense shadow of the full stomach. Only a small quantity (3 to 4 oz.) is therefore given at first. This can be displaced upwards by manipulation and smeared over the interior of the stomach without difficulty. Another 6 oz. or more may be administered later as required, especially if the whole intestinal tract is to be examined.

#### NUMBER OF EXAMINATIONS.

Owing to the increasing number of cases requiring the opaque meal examination, everything not proved of diagnostic value has to be discarded. For instance, after carrying out a few tests, I found that frequent examinations, to ascertain the rate of emptying of the stomach, were of little diagnostic value, as the rate of emptying is influenced by many unknown factors.

If some mechanical narrowing is present, such as of the pylorus or duodenum, which is sufficient to cause delay, this should be easily detected at the first examination. Nothing further is learnt by hourly noting the quantity of the meal which has passed into the small intestine. In normal stomachs, the meal should be seen passing through the duodenum at the first examination. Carman states that free opening of the pylorus is retarded by coarse particles of food.

#### IMPORTANCE OF CONCENTRATION OF ATTENTION.

Concentration is essential in examination of the intestinal tract. Outside disturbances should be avoided. Departmental organization should run smoothly and there should be no delay between examination of patients.

#### SYSTEMATIC EXAMINATION.

Complete protection to the operator from rays being impossible whilst screening, fluoroscopic work should be carried out expeditiously. Examination should be systematic. In examination of the thorax, for instance, some definite order or routine should be followed; examine the diaphragm first, then the heart and aorta, apices of lungs, hilum and lungs themselves, and so on. There is no necessity to return to a section passed as normal. The same systematic method should be employed in all fluoroscopic work. However obvious a lesion, this routine examination should be persisted in. The presence

of a chronic gastric ulcer, for instance, does not exclude the possibility of a duodenal ulcer.

#### APPARATUS.

In all radiological work, apparatus should be as simple as possible. This is still more important when radioscopy palpation is being employed. The chief features of an upright diascope should be:—

(1) Operator should sit comfortably and apparatus so arranged that he is not cramped when palpating patient's abdomen.

(2) If the latter point be observed, operator has full control of patient's movements. Hips can be gripped and patient moved in any direction without verbal instructions.

(3) The best fluorescent screen should be used for gastric work. It should be small; it need not be larger than 10 in. by 8 in. Palpation is easier under these conditions and unnecessary fluorescence around the illuminated area avoided.

(4) Patient should have firm support against which to lean his back, as full palpation cannot otherwise be used. For this purpose, I have a metal cylinder, about 8 in. long, fixed to the face of the diaphragm.

(5) There must be sufficient space between back of screen and front of patient for the hands to palpate.

(6) There should be an arrangement for placing a cassette in position if a radiograph is required. I have carried out this by placing a loaded cassette to the right and left of the screen—protected from the rays—ready to be slipped one after another behind the screen at any moment.

*Protection.*—Protection may be considered as part of the apparatus. Thick lead around tube is the safest protection. Lead face holding the diaphragm should be extra thick and the diaphragm shutters made up of iron and lead. Two sheets of best tested (N.P.L.) glass should be placed over the screen. Under no circumstances should it be possible for rays to be projected off this area. An aluminium filter over the diaphragm opening is sufficient to protect the patient. For radioscopy palpation, I use specially made protective gloves—light and supple, with protective material on front and back and lined with soft fabric.

In screen work, the following protective rules should always be observed:

(1) Distance between tube and screen should never be less than 30 in. (2) Use minimum amount of current, increasing it for a few seconds as required. (3) Examination should be carried out as expeditiously as possible.

Before new apparatus is used, protection under all working conditions should be thoroughly tested with a fluorescent screen or other means, from floor upwards. Efficient earthing should be seen to. Under these conditions, the method here described is perfectly safe.

#### POSITION OF PATIENT.

Unless the diascope is designed for radioscopy palpation as explained above, the patient will have to be examined both in the upright and horizontal positions, the latter being used chiefly to visualize the pylorus and duodenum.

With a properly designed diascope, I find that a much better view of the pylorus and duodenum can be obtained when the patient is standing. The meal is manipulated more easily into the pyloric end of the stomach, and the low position of the pylorus and duodenum when the patient is erect simplifies palpation. If, however, the stomach is much dilated, the contents are difficult to control by palpation, and the right horizontal position has to be resorted to, before the pyloric end can be satisfactorily filled. The mechanism for moving the

tube under the couch should allow both hands to be free for palpation. I use a foot stirrup for the moving tube box. To obtain good views of the pylorus and duodenum, the patient should be lying on his right side. The degree of tilt varies with different patients. In some cases, the best view is obtained with the patient flat on his face.

Success in this method of examination is, to a great extent, dependent on the correct form of palpation being used. For instance, if the abdomen is prodded with the tips of the fingers, efficient palpation is rendered difficult, owing to the resistance set up by the abdominal muscles. It is important, therefore, that pressure should be applied with the *flat of the fingers* and not with the tips. The hands must be trained to work in conjunction with each other. The whole proceeding must be carried out gently and with persuasion. Rough handling is to be avoided—in fact, in some cases, it may be dangerous to the patient.

The examination in detail through its various stages is as follows: The patient, with a special garment, is standing up in the diascope, with his back to the tube, holding the handles on either side for support. The barium cream in a suitable container is placed on a small shelf close to his left hand. The patient is instructed beforehand that he will be told when to drink it. It is more satisfactory if the patient holds the cup in left hand, as the first part of examination necessitates his turning to the left, and if the right hand is used the cream may be spilt.

The operator, comfortably seated in front of the screen, grasps the patient's hips, and commences examination with a rapid but systematic survey of the thorax and abdomen. Patient is given a half turn to his left by means of the hips, bringing him into the oblique position, thus opening up the posterior mediastinum. He is then told to drink, and the meal is carefully followed in its passage from mouth to stomach, as the œsophagus or thorax may be the site of the suspected gastric lesion. The meal settles in few seconds and fills the most dependent part of stomach. Palpation should not commence until this has taken place. The type of stomach, position, &c., is quickly noted.

Whatever quantity of meal is given, without manipulation the cardiac end of the stomach cannot be distended to the same extent as the lower segment. This is why many small ulcers high up on the lesser curvature are missed. To overcome this disadvantage, with the patient still in the oblique position, firm pressure is exerted on the lowest part of the stomach with the flat of the left hand—the left side of the patient's back pressing firmly on the compressor cylinder fixed on the front of the diaphragm.

The meal is now seen to be displaced upwards until the upper segment of the stomach becomes fully distended and any defect in outline can easily be noted. A small ulcer may be lurking on the anterior or posterior surface of this section, when it would be masked by the meal shadow. To overcome this difficulty, pressure of the left hand is relaxed slightly and the right hand is brought into play by working in conjunction with the left; the meal can thus be displaced from any part of the stomach at will.

The process is not easy to describe; it is comparable to whitewashing gastric rugæ. The ulcer crater in their midst would be noted to hold white-wash more easily than the surrounding folds of mucous membrane, a fleck or dark spot among distorted rugæ being the result, and if persistent, would be typical of an ulcer.

If small ulcers are not to be missed, every inch of stomach wall must be examined in detail. If the organ is too full this cannot be done, as it is difficult or impossible to displace the contents sufficiently. It is better, therefore, to start with a small quantity of cream (3 oz.) giving more later, if required.



Both hands work together from the cardiac end, filling and emptying each segment in turn. As the pyloric end is approached, the patient is gradually brought round from the left oblique position until square with the operator. This opens up the pyloric end, and in most cases a slight turn to the patient's right gives the best view of the pylorus and duodenum, but there is no orthodox position; patient should be moved in whatever direction gives the best view of the area under examination. The pylorus is now reached and the filling of the duodenal cap carefully observed. This takes place fairly rapidly if palpation has not been too energetic, in which case spasm of the pylorus is likely to cause a short delay. Light massage soon dissipates this.

The duodenal cap should be examined in the same systematic way. This is simple if it stands away from the shadow of the pyloric end, but in some cases it gets tucked away behind and it is difficult to find the position giving a clear view. Great care is necessary in examination of the cap, as small ulcers are easily overlooked, the defect in the shadow in some cases being extremely small. Inspection of the duodenum completes the examination.

The rate of emptying is of no diagnostic value. Frequent examinations are thus unnecessary, unless as confirmation of some abnormality. Occasionally the meal passes rapidly out of the stomach; this necessitates pressure over the pylorus while the cardiac end and fundus are being examined, pressure being relaxed as the pylorus is approached. Once the meal is in the small intestine, the stomach examination should cease.

Radioscopy has not developed to the same extent as radiography, which is a pity. I hope that this paper will stimulate other workers to rectify this, as I think in future years it will be found to be the most accurate method of diagnosis.

## Some Effects of Exposure to Radium upon the Alimentary Canal.

By J. C. MOTTRAM, M.B.

(From the Research Department, Radium Institute, London.)

It has been recently observed that exposure of dogs to large doses of X-rays quickly produces desquamation and necrosis of the intestinal mucosa [1]. Barlow exposed various animals to the gamma radiation from 5 gm. of radium, and found as an early change, desquamation of the intestinal mucosa, and mucoid degeneration in the case of the colon and rectum [2].

The following observations have been made to discover what are the first histological changes in the alimentary canal, and at what period after radiation they occur, with a view to seeing whether they could in any way account for some of the late effects of exposure to radium, for instance the thrombopænia and anæmia.

Six rats were exposed continuously to 220 mgr.  $\text{RaBr}_2 \cdot 2\text{H}_2\text{O}$ , distance 8 in., screen 0.1 mm. lead plus 0.12 silver. They were killed with gas after periods of one, two, three, four, five and six days' exposure. Six similar non-irradiated rats served as controls. All histological procedure was carried out in duplicate.

The following tissues were examined: Stomach (cardiac and pyloric portions), small intestine at distances of 5 in., 10 in., 15 in., 20 in. from the stomach, cæcum, and colon at distance of 2 in. from cæcum.

Numerous staining methods were used, more especially Van Gieson—Nile blue for bacteria and carbol-thionin with iodine for mucus.

[January 19, 1923.]



The changes in the cæcum are first described. In this region of the alimentary canal the mucosa consists of a single layer of columnar cells, everywhere pitted with short mucous glands, which project into a loose connective tissue. No deviations from the normal were observed after twenty-four hours' exposure. The first changes were found after forty-eight hours, and consisted in a very great increase of mucinogen granules in the cells lining the tubular glands, the granules occupying clear vacuoles in the cell protoplasm. This granule formation and vacuolation occurs in the normal goblet cell, and is the first stage in the formation of mucus. In the radiated specimens it is, however, not confined to goblet cells, but columnar cells also show the change; moreover, it occurs in an extensive and disorderly manner. It may be described as the first stage of a mucoid degeneration rather than the exacerbation of the normal production of mucus.

After three days the granular degeneration is still present, with the difference that the granules tend to be larger and less numerous, and the hyaline material more abundant. Normal goblet cells are rarely seen except in the discharged condition. The mucoid degeneration involves the cells at the base of the glands, where goblet cells are not usually seen, so that a striking difference is now found between experiments and controls.

After four days' exposure very few mucinogen granules are seen. Normal goblet cells are not now to be found. The mucous material in the radiated specimen stains much less strongly pink with thionin than normal goblet cells. Except at the base of the gland there is now no mucus occupying the lumen of the gland, or plugging its mouth.

Apart from this mucoid degeneration no other histological changes were seen, except that the columnar cells stained somewhat more faintly than the normals and their nuclei were larger and less deeply stained. Mitotic figures were as numerous in the experimentals as in the controls.

In the fifth and sixth day specimens little further change occurs: no further mucus is produced, and that which is found at the base of the glands stains only faintly pink with thionin. Small areas of desquamation of the columnar cells, leaving bare the connective tissue, were occasionally observed.

The changes in the colon were identical with those in the cæcum, except that in the fifth and sixth day specimens no mucus staining material was found, the gland being filled with a hyaline material which did not stain pink with thionin, nor brown with iodine, so that this degeneration may be described as pseudo-mucoid rather than mucoid.

In the small intestine similar changes occur wherever mucoid-producing tissues occur. The mucous glands of Lieberkühn are especially involved, and here the mucosa between the villi tends to become desquamated. In the stomach no changes were observed; neither in the pyloric nor cardiac portions do mucoid glands occur.

One other change was observed: in the case of the cæcum and colon in the fifth and sixth days specimens the intestinal contents were found post mortem to be closely adherent to the mucosa, whereas in the controls they were very easily separated. This change was also seen in the histological specimens; in the experimental specimens of the fifth and sixth day bacteria were found everywhere closely adherent to the mucosa, and in many places invading the epithelial cells and the connective tissue beneath. Protozoa (*trichomonas*) were also found invading the cells of the mucosa in the radiated specimens.

Summarizing these findings it may be said that at first there is an excessive production of mucus in an abnormal manner, a mucoid degeneration.

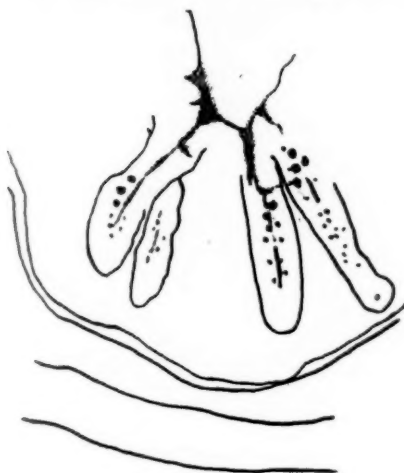


FIG. 1.—Control.

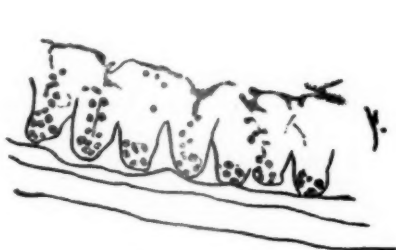


FIG. 2.—0.15 rads.

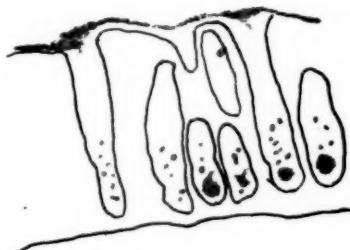


FIG. 3.—0.25 rads.



FIG. 4.—0.4 rads.

tion, and later a stoppage of mucus production, accompanied by a pseudo-mucoid degeneration of the cells. The absence of mucus allows the bacteria of the colon and caecum to come into intimate contact with the mucosa, and this is followed by a bacterial invasion of the epithelium and connective tissues.

The diagrams reproduced (*see* figs. 1-4, p. 43) illustrate the changes in mucus production of the caecum. They are low-power camera lucida drawings of specimens stained with iodine and thionine blue. After 0.15 rad there is, as shown, an increased production as compared with the control, and especially at the bases of the glands, where normally mucus is not formed. Subsequently the mucus production ceases, so that after 0.4 rad only a little mucus is to be seen at the base of the glands.

The question as to whether these effects of exposure to radium can in any way account for the thrombopænia and anæmia which occur later may now be considered.

The thrombopænia begins under the same conditions of exposure on the fourth and fifth day, but is not profound until the tenth or twelfth day [3], so that its beginning corresponds in respect of time with the bacterial invasion of the mucosa. Now it is known that the introduction of bacteria into the circulation causes a profound thrombopænia, so that it may well be that this change in the mucosa may condition the thrombopænia. On the other hand, the fact that radiation produces a rise as well as a fall in platelets suggests that it may be the primary cause in both cases. As regards the anæmia, this occurs a little later, and may also be due to invasion of the blood stream by bacteria from the alimentary tract: though Møller [4] is of opinion that the anæmia is the result of hæmorrhages resulting from the profound thrombopænia which follows radiation; on the other hand, there is evidence that infection may be an important factor [3]. The necrosis and desquamation of the villi in the small intestine found by Barlow [2] after large doses of radium, and by Warren and Whipple [1] after large doses of X-radiation, follows the bacterial invasion of the mucosa, and is probably due to this infection.

These findings show that the normal production of mucus in the alimentary tract is interfered with by very small doses of radiation, less than would be required, for instance, to effect histological changes in the testes or skin. It follows that when treating patients with large doses of radiation care should be taken to expose the alimentary canal, and especially the lower portions, as little as possible. This may also apply to the nasal passages, trachea and bronchi, and in fact wherever mucus is secreted. The symptoms which are likely to occur if efficient protection is not given are mucoid diarrhoea, general symptoms of toxæmia from absorption of intestinal bacteria, and later blood changes such as thrombopænia and anæmia, due to a bacterial invasion of the blood.

The radium used in this investigation was on loan from the Medical Research Council.

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## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

### The Cauterization of Adhesions in Artificial Pneumothorax Treatment of Pulmonary Tuberculosis under Thoracoscopic Control.

By H. C. JACOBÆUS, M.D.<sup>1</sup>

*(From the Medical Service II of the Serafinerlasarett, Stockholm, Sweden,  
Professor H. C. Jacobæus, Director.)*

FOR about ten years I have been occupied with endoscopy of the serous cavities, peritoneum and pleura. At first, I was interested only in the diagnostic advantages which could be gained by such a method. In a case of ascites, after tapping and replacing the fluid by air, I could by endoscopy get a clear view of the abdominal organs. There was thus no difficulty in diagnosing liver cirrhosis, malignant tumour, Pick's disease, syphilis, &c. Further, in carcinoma and tuberculosis of the peritoneum, I could see changes characteristic of these diseases. After first performing endoscopy, or laparoscopy, only on patients with ascites, I have, during the last few years, to a larger extent, also carried out the examination on patients without ascites and thereby widened the sphere of the method considerably. I have further combined laparoscopy with simultaneous X-ray examination of the abdominal organs, with the air still left in the abdominal cavity, performing the latter according to the methods of Long, Weber and others. These methods of examination complement each other in a very successful way, especially with regard to disease of the liver and the spleen and the formation of adhesions in the abdominal cavity. It is not yet possible to judge how great a value they may obtain in practice.

Without doubt, the predominant value of endoscopy centres around the examination of the pleural cavities, the so-called thoracoscopy. With regard to the chest cavity there is, as we know, nothing corresponding to exploratory laparotomy. Further, thoracoscopy is so simple a method that it can be performed without inconvenience in every exudative pleurisy which is available to a thoracocentesis. The ocular examination of the pleural surfaces is in most cases relatively complete. In cases of so-called idiopathic pleurisy I have also succeeded in finding distinct tubercles in most of them. In the differential diagnosis of tumours and pleurisy of other origin, thoracoscopy is likewise of no small value. After some practice it is at least possible, with some degree of certainty, to differentiate between tumour metastases and tuberculous changes. In doubtful cases one can by test-excision, under guidance of the

<sup>1</sup> At a Joint Meeting of the Section of Electro-Therapeutics with the Roentgen Society, at Manchester, November 17; also delivered as an Occasional Lecture at the Royal Society of Medicine, November 22, 1922.

thoracoscopy, decide the nature of the pleurisy in the special case. Even solid intrathoracic tumours can be observed by thoracoscopy, and their relations to neighbouring structures, lung, thoracic wall, &c., be determined much more clearly than by any other method. By this an evident practical advantage for an intended operation is gained. As we shall see further on, this is its principal use in major surgery.

The second, and, from a practical point of view, the most important field for the use of thoracoscopy comprises those surgical operations which can be performed directly under guidance of this method. By thoracoscopy in the pneumothorax treatment of lung tuberculosis an especially fine picture of cord or membrane-like adhesions between lung and thoracic wall is obtained. This led me to try to work out a method under the guidance of the thoracoscope of removing such adhesions as impede the treatment. It is a well-known experience that a single cord-shaped adhesion, which attaches the lung to the thoracic wall and thereby prevents collapse of a cavity, may lead to the failure of the whole treatment in artificial pneumothorax. A recently published paper by Gravesen, from Professor Saugman's sanatorium, contains the following tables which prove the injurious results of these adhesions:—

TABLE I.—CASES WITH COMPLETE PNEUMOTHORAX WITHOUT ADHESIONS.

(Three to thirteen years after discharge.)

Able to work	...	...	...	23 = 70·2 per cent.
Not able to work	...	...	...	1 = 2·1 "
Died from tuberculosis	...	...	...	11 = 23·4 "
Died from other causes	...	...	...	1 = 2·1 "
Unknown	...	...	...	1 = 2·1 "

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TABLE II.—CASES WITH COMPLETE PNEUMOTHORAX BUT WITH LOCALIZED, EXTENSIVE ADHESIONS.

Able to work	...	...	...	14 = 33½ per cent.
Died from tuberculosis	...	...	...	28 = 66½ "

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TABLE III.—CASES WITH INCOMPLETE PNEUMOTHORAX WITH MORE OR LESS EXTENSIVE ADHESIONS.

Able to work	...	...	...	5 = 11·1 per cent.
Died from tuberculosis	...	...	...	39 = 86·7 "
Died from other causes	...	...	...	1 = 2·2 "

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The injurious influence of the adhesions is plainly demonstrable from these tables, which also give one an impression of their frequency. I cannot here enter into the different methods attempted by others for the removal of such adhesions. I can only say that none of them has attained any practical importance.

As it was rather easy to observe the above adhesions by thoracoscopy, the idea occurred of cauterizing them by introducing a galvanocautery through another opening, under the guidance of the thoracoscope. My first attempts were made in 1913, and since then I have performed, altogether, seventy-five such operations. The operation has been further performed in more than thirty cases by Saugman; of these his assistant, Gravesen, has published sixteen. Twelve cases have been published by Holmboe and twenty more cases by Skjærgård and others, six by Sömme, six by Begtrup-Hansen, three by Christoffersen, and ten by Dahlstedt. To date, certainly about 200 operations

have been performed. To obviate the opprobrium that must follow failure in performance one must ascertain what cases are likely to succeed under the method and to reject those in which the operation may prove difficult or perhaps dangerous.

#### TECHNIQUE.

The preparations for the operation need not be very extensive. It is not necessary to give the patient morphine beforehand. If he is very anxious I find it better to give him bromide, luminal or similar remedies. One patient, in my experience, who was given morphine, had an idiosyncrasy for it, so that during or after the operation that patient became sick and the act of vomiting set up a severe cutaneous emphysema, which developed from the puncture opening in the chest wall.

It is of the first importance to be sure that the electrical apparatus is in perfect working order.

Immediately before the operation my practice is, after the method of Saugman, to make a fluoroscopic examination and draw the points of attachment of the adhesions and their extensions on the patient's chest. This is a very good guide to the place for introducing the thoracoscope, it being always most advisable to have the light as near the field of operation as possible.

The patient is placed on his healthy side with a large, hard roll-pad under the chest as near the arm as possible, and a smaller pillow under his head. The reason for this posture is to get the pneumothorax half of the chest as convex as possible. It is also advisable to let the patient's arm on the operated side lie upward and forward, so that the scapula is pulled upward and outward. The chest wall gets thinner, the interspaces are more easily felt, and the local anæsthetic can be given with more assurance if the thoracoscope can be introduced higher up on the chest wall without difficulty.

At the operation proper I first introduce the trocar for the thoracoscope after a careful local anæsthesia, generally one to three interspaces below the level where the adhesions are marked by means of the fluoroscope. This is of course not possible if the adhesions are localized to the apex, but one has to try to find an interspace as high up in the interscapular area as possible. The higher up on the chest wall one gets the denser are the muscles and the more difficult it is to make the anæsthesia quite effective.

The thoracoscopy, which is first performed, aims at giving as complete an insight into the conditions in the pleural cavity as possible. If it is a matter of single cord-shaped adhesions, then it is rather easy. In introducing the thoracoscope rather far back near the spine, before the denser dorsal musculature begins, one gets the longitudinal direction of the lung at right angles to the thoracoscope. In this way it is possible to get a survey of the lung in its longitudinal direction. As the adhesions generally extend from the lung to the chest wall at a more or less wide angle, it is evident that with this direction of the thoracoscope and the field of vision it will be easy enough to find and localize the adhesions in question. If the adhesions are membranous the difficulties are greater and no rule can be established, but after some practice it is in most cases possible. The same is the case if the lung is broadly attached to the anterior or posterior chest wall and also with narrow adhesions in different directions. Only experience can solve the difficulties in such cases.

I generally introduce the trocar for galvanocautery into the lateral region, mostly perhaps in the anterior axillary line. In this way the galvanocautery is inserted in a direction parallel to the longitudinal direction of the



lung and chest wall. It is desirable to be able to move the galvanocautery along the inside of the chest wall as extensively as possible. This is best attained by an incision in the lateral region where, on the whole, with regard to posteriorly and anteriorly fixed adhesions, it can be done within a less restricted area further back. With regard to adhesions which extend directly laterally, it is better to enter a little more forward, for example, in the anterior axillary line. If one enters the lateral region, it may easily happen that in handling the grip of the electric burner one's movements are hindered by the patient's hip bone.

The other stage of the operation, the cauterization itself, is then begun. It ought to be carried out with quite a weak glow, and slowly. Its difficulty is intimately dependent on the thickness and the extension of the respective adhesions and on the indications upon which the operation may be based. I, therefore, pass on to the description of the operation itself.

The galvanocautery is carefully introduced in the direction in which the adhesion is presumed to be situated. With some experience this is fairly easy of accomplishment, and I have many times introduced the cauterizer directly on to the adhesion which I desired to burn off. If this does not immediately succeed, the adhesion has to be found with the thoracoscope in the thoracic cavity which, to begin with, may give rise to certain difficulties. After this, the platinum point should touch the adhesion at the spot selected for cauterization. As a general rule, with cord-like adhesions, I perform the cauterization where the adhesion is narrowest. If a cavity is visible on the X-ray film, immediately below where the adhesion is fixed to the lung, the cauterization should be done as close as possible to the chest wall. Technically this is slightly more difficult, but it causes the patient considerably more pain. In these cases the patient has a real sensation of a burning pain which is diffused throughout the whole half of the chest. Where apex adhesions are present the pains radiate to the arm. If the cauterization is performed at a little distance (2 to 3 cm.) from the chest wall, the patient has scarcely any sensation of the cauterization, but has mostly a disagreeable feeling of traction on the adhesion, caused by the pressure of the galvanocautery.

During the cauterization itself one has to decide the intensity of the glow of the galvanocautery. This is of great importance because of the possibility of hæmorrhage. After switching on the current, it ought to take five to ten seconds before the platinum needle becomes aglow, and then there should be only a red glow. A white heat is absolutely contra-indicated. Sometimes I have allowed the platinum needle to glow in the pleural cavity and then applied the glowing needle to the adhesion. At other times I have first placed the needle over the adhesion and then switched on the current. The difference is that in the latter case one obtains a considerably slower heating of the needle. There is no great difference between these two methods.

The cauterization should proceed very slowly. In slightly pressing the galvanocautery against the adhesion one feels how fibre after fibre is loosening. Then, bit by bit of the adhesion can be burnt off, until at last it is completely severed, when the lung collapses by its own weight and usually a space of several centimetres appears between the two burnt off stumps of the adhesion.

As above mentioned, the danger of cauterization is hæmorrhage. On the one hand, it is possible to encounter such large vessels in the adhesions that even under cautious cauterization hæmorrhages occur. This difficulty has scarcely been solved. Forlanini and Permin state that upon section they have found veins the thickness of a quill in adhesions and on the pleural surfaces.



I, myself, have seen varicose veins on the surface of the adhesions but not of such thickness. Not infrequently I have burnt off such small veins without hæmorrhage. Whether the larger veins also imply any more serious danger of hæmorrhage, I am, of course, unable to decide from my own experience. In my opinion these vessels are not the most dangerous. The pressure in them is certainly very low, and if, in case of hæmorrhage, a saline solution is injected in sufficient quantity, the hæmorrhage should in all probability cease before it becomes dangerous to life.

It is my belief that it is only when an artery is burnt right through that hæmorrhages really dangerous to life are to be expected. Whether such are to be found in the adhesions is also not fully clear. The only microscopic investigations of cord-like adhesions, which have been made to any large extent, are those which I carried on some years ago. In them are to be found vessels, the nature of which cannot be decided. They have a very thick musculature; so thick that sometimes obliteration ensues. Cauterization of such vessels surely does not cause any hæmorrhage dangerous to life. The retraction of the adhesion which sets in after the cauterization should certainly entail a further shrinkage and probable closure of the vessel.

The danger of a very serious hæmorrhage arises if during the cauterization the lung is penetrated and an artery severed. Here there is neither any hypertrophy of musculature of vessel wall nor is there any considerable retraction of the surrounding tissue to be expected. This is accordingly the principal danger which threatens in the field of operation.

Only once have I experienced hæmorrhage of any consequence. In this case there was quite a narrow thread-like adhesion. The cauterization was carried out very easily and quickly, in a fractional part of a minute; but afterwards blood escaped rather rapidly in drops from the parietal stump of the adhesion. In all, the hæmorrhage might be estimated at the most to have been about 200 c.c. My impression is that there was too strong a glow of the galvanocautery. Other authors, such as Saugman and Skärgård, have at times had smaller hæmorrhages, which were considered to be caused by too strong a glow at least by the latter operator.

Only once in all the operations known to me (so far in more than 100 cases), has a fatal hæmorrhage ensued. In one case, where the operation, performed by Dr. Dahlstedt, proceeded quickly, a severe hæmorrhage suddenly set in. The blood spurted into the thoracic cavity. The prism of the thoracoscope became constantly splashed, and it was therefore impossible, under its guidance, to get at the bleeding point with the galvano-cautery, in order to stop the bleeding by means of a fresh cauterization. The bleeding became so severe that the patient lost consciousness. Things began to look very threatening. The very circumstance that the burning immediately before the catastrophe proceeded so quickly, is an indication that too strong a glow of the platinum needle had been used. In another case, where the operation was performed by Skärgård, a similar arterial hæmorrhage arose from the lung. Here the operator succeeded in stopping the bleeding by continuing to burn the bleeding part proximally. This method is indeed very daring, but it partly shows that the hæmorrhage really comes from a pulmonary vessel and partly that mere chance is probably a causal factor because it is more likely that the bleeding vessel was still thicker at the last cauterization. I myself have stopped quite small hæmorrhages by holding the galvano-cautery close to the bleeding point. Should a large arterial hæmorrhage arise I think it best to get as high a pressure as possible in the pleural cavity in question. The best

method to obtain this end should be to fill it up with saline solution, which ought to stop the hæmorrhage.

My conception of the cause of the hæmorrhage and its prevention, is that—

(1) *The severer hæmorrhages probably, and, if so, principally arise from cauterization of the lung itself.*

(2) *They can most effectually be prevented by using a weak glow and by burning as slowly as possible.*

In previous papers I have, from a practical point of view, classified the adhesions into three different groups: (1) Apical, (2) lateral, and (3) diaphragmatic. The limit between at least the first two classes is rather vague, and I have, therefore, fixed the second rib as the line between them.

The commonest belong to Group 2, the lateral adhesions. Of my fifty cases, forty-five belong to this group. Some of them border on Group 1, where we thus find the largest and thickest adhesions below the second rib and some smaller cord-like ones higher up in the dome of the diaphragm itself. Or we find membranous adhesions, which continue toward the apex above the second rib. The reason why I have separated an upper group is on account of the more difficult technique which is required for the apex adhesions. The technique of these cauterizations has for its object the introduction of the thoracoscope as high up as possible in the back, after the scapula has been pushed upward and forward. Others, for example, Skärgård, have found it suitable to insert the thoracoscope in front or in the axilla. It is possible that this technique is convenient for certain cases of adhesions to the anterior part of the chest wall, but I have no experience of this.

The difficulty in cauterizing these adhesions lies in the following circumstances. Most cavities exist in the apex of the lung, and these are attached to the dome of the diaphragm itself by short adhesions. The mobility of the lung in respiration is here rather limited, and from this it follows that these adhesions during life are exposed to relatively little tension and are therefore short, and consequently there is a risk of burning the lung. At all events, it is necessary to burn as close to the chest wall as possible, which often causes this operation to be rather painful. In this group I have had two peculiar cases, in which the operation went well and the condition of the patient obviously improved, but after one or two months the patient suddenly fell ill with acute empyema, in the one case with mixed infection. The question arises whether the cauterization of the rather large adhesions has had any unfavourable influence, and whether a rupture of the lung has taken place in some weak point, where the burning has been performed, or whether it has set in quite spontaneously. I cannot give any answer to this, as I have not had the opportunity of seeing any autopsy. I wish merely to draw attention to it.

The lateral adhesions from the area below the second rib to the diaphragm are the most numerous. They are mostly quite easy to get at. If they are attached wholly in the lateral region, difficulties may sometimes arise; in this respect, that if the burner is also introduced into the lateral region, the hip bone may, as already mentioned, get in the way of one handling the burner with one hand. In such a case the best to do is to introduce the trocar for the burner either further forward or further backward.

Diaphragmatic adhesions also deserve closer consideration. In all my cases of this kind it has happened that the lung above has been attached to the chest wall to such an extreme degree that it has not been possible to undertake any cauterization there. Besides, there have been present narrow, cord-

shaped adhesions to the diaphragm—sometimes only one, sometimes many. By burning them off it is possible to get the lung pushed up and thereby also to obtain a more complete collapse of the lung. From a technical point of view, the cauterization presents the difficulty that the patient must hold his breath during the cauterization, because otherwise the adhesions are in constant movement. If the cauterization succeeds in a single burning there is no difficulty; but if, as is generally the case, the galvanocautery has to be applied several times on this swinging band it can be ever so troublesome technically to remove such adhesions. One advantage is that the cauterization of such adhesions is completely painless. Another question is the practical use of such cauterizations. In my opinion, it is only in exceptional cases that there is any real benefit to be obtained by them.

#### INDICATIONS.

It is necessary before each cauterization to have an X-ray photograph of the collapsed lung with its adhesions. The first question that arises is, then, how much can be judged from the X-ray picture as to the prospects of a successful operation. In cases where only one or two isolated, cord-like, long adhesions are to be found, the indications are easy, and one can assume at once that from a technical point of view it is possible and very likely easy to cauterize the adhesions. It is more difficult to decide those cases in which the X-ray apparently shows too thick adhesions, or in which the surroundings are quite indistinct and one does not know the nature and extent of the adhesions. In the latter case one has to rely on the thoracoscope. In some cases the X-ray picture has been misleading with regard to the possibility of cauterization, for from it one obtained the impression that the lung was held expanded by a thin membrane, which could easily have been burned off. In one case the lung was broadly attached to the posterior chest wall, and what looked like an adhesion was only a protruding fold from an area where the surfaces were matted together. The cauterization in the latter case consequently did not succeed. This event ought to be discovered, or at least suspected, if an X-ray picture is taken both from front and back. More and more have I found that if seemingly cord-shaped adhesions exist near the anterior or posterior chest wall they are seldom free, but are attached to the chest wall throughout their length. The result of cauterization in such cases has been only the removal of the more protruding folds, while the larger part of the adhesion remained.

Undoubtedly the best procedure in such cases is stereoscopic X-ray examination, which has been practised in our hospital only a few weeks. This method should also be of the greatest importance in the localization of other adhesions and their disposition in the pleural cavity. I believe, however, that in one respect its value is subject to certain limitations, inherent in the ordinary X-ray picture, which usually shows too few adhesions. I can state that in the majority of the cauterizations there have always been found more adhesions than were visible on the X-ray picture. This is generally of less importance, because the unobserved adhesions are, as a rule, so thin that they do not present any serious obstacle to cauterization.

Experience shows, however, that cauterization in most cases is more difficult than would be expected from the X-ray picture. In one of my cases the X-ray showed very different conditions from what were actually found. It was a case of a dense adhesion at the apex sent to me for cauterization. From the X-ray photograph cauterization seemed possible, but thoracoscopy made it

evident that the adhesion was so dense that I would not even attempt cauterization. The patient was referred to Dr. Key for a plastic operation of the thorax. On account of the apparently limited thickness of the adhesion, visible by X-ray, Dr. Key attempted thoracotomy in the first interspace, with dissection of the adhesion, as in a previous case, published by us jointly. But the adhesion was too dense even for such an operation, since it filled nearly the whole apex. Later, a thoracoplasty was performed. This case is not unique. Both from the literature and from private information I have become familiar with cases in which attempted thoracotomy has shown the adhesions to be much thicker than was originally supposed. In these cases the operation was consequently more difficult and involved more extensive interference than had been anticipated, and in the majority it had the very serious sequel of tuberculous empyema, with or without mixed infection.

The other method of determining the possibility of operation is thoracoscopy. This will, according to my opinion, become more and more important in deciding the possibility of operation in the more serious cases, not only with regard to cauterization but also to other surgical methods.

By thoracoscopy one gets a perfectly accurate picture of narrow, cord-like adhesions, up to the thickness of a small finger. The difficulties appear in the case of membranous adhesions and those attached to the surface.

In perhaps the majority of cases one can see the compressed lung in its proper colour, greyish-blue with coal pigment, while the adhesion is greyish-red with distinct vessels in its longitudinal direction. The tissue is loose and spongy when pressed with the galvanocautery. Often the adhesion runs like a membrane along the ribs. It vibrates when the patient coughs or is shaken. In these cases there is also a transparent bluish colour. Without doubt, there exists an organic, membrane-like granulation tissue, which has become stretched and forms the essential adhesion. These adhesions are the most favourable for cauterization, which may be done easily, rapidly, and without pain worth mentioning. The danger of hæmorrhage surely cannot be great. I have burnt off adhesions with rather large, even varicose vessels without any bleeding whatever. With some experience it is not difficult to see the difference between the lung and the adhesion.

In other cases one sees the greyish-blue lung, with its coal pigment, stretching right out to the pleura, possibly with a small membrane, resembling a pleural membrane, in the fold between the lung and the chest wall. In these cases the thing to do is to loosen the pleura around the point where the adhesion is attached. If it is attempted to continue the cauterization, the distance between the lung and the chest wall gets smaller and smaller, and it becomes really impossible to continue without penetrating the lung. In such cases I have loosened the tissue around the adhesion as much as possible and then continued with pneumothorax treatment under high pressure. After an interval of time I have also performed a new cauterization in the same way. In one case I succeeded the second time in burning off an attachment, which proved impossible at the first attempt. I think that it is in this way that one can, may be in a limited number of cases, enlarge the scope of the method. I performed the operation in two or more stages in only four of my first forty cases, using a moderate over-pressure between the two stages. The essential reason for this procedure is that it enables me in still higher degree than before to avoid burning the lung. I think this point is very important. In such cases, where the lung

is directly attached without this intervening tissue, I do not think it advisable to force any cauterization.

It is these latter cases, in which the adhesions in greater part are formed by lung tissue, which most often complicate the cauterization. In such cases one has to expect partly, as already mentioned, the existence of larger vessels, and partly tubercular changes and offshoots of cavities. The difference between this type of adhesions and the preceding one is not always distinct; there are transitional forms with both granulation and lung tissue, as proved by histological investigations which I have made. The cord-like, tense adhesions, thinner than a pencil, may well contain lung tissue and perhaps also offshoots from cavities, but nevertheless, in my opinion, they are not dangerous to burn off; just as in the case of hemorrhages, one has to attach a certain importance to the immediate, marked shrinkage of the adhesion after cauterization in preventing tuberculous infection of the pleura. While working with thoracoscopy, one can see the adhesion stumps shrink to half of their original size or less, within a few minutes. Without doubt, where offshoots from cavities exist they must be compressed and obliterated. This sounds bold and hypothetical, but I believe that it is so. If these adhesions are microscopically examined one not infrequently finds offshoots from cavities filled with necrotic tissue. To date, several hundred adhesions have been burnt off, and in only one known case (Holmboe) has a cavity been opened, with resulting mixed infection in the pleura. In this case there was a surface adhesion of considerable thickness, and the operation had been going on for nearly two hours without complete success in burning off the adhesion. The patient was so well on the second day after the operation that a new thoracoscopy was performed, but then quite unexpectedly there occurred a sudden perforation of the lung with mixed infection and acute empyema, which in a few days proved fatal. The autopsy did not entirely reveal how the infection got into the pleura or how extensive the cauterization had been. In making this statement it is not my intention to deny that the result of the operation was unhappy. The incomplete cauterization in the case in question was perhaps of importance, because the shrinkage which occurred could not have resulted to the same degree from any other cause. The tedious and severe pleuritis after the operation seemed to be caused by the opening of tuberculous foci by the cauterization, and then the infection spreads to the pleural cavity.

I have not infrequently had cord-like adhesions, which at X-ray examination and on thoracoscopy have been technically easy and possible to burn off, but in which I have nevertheless not done this, because it was necessary to consider the clinical effect obtainable by the removal of the adhesions in question. According to Saugman's and Gravesen's statistics, the difference between the prognosis of those cases with and those without adhesions is so great that practically all cord-like adhesions, which are technically possible of removal, provide an indication for cauterization. I believe that as long as one cannot, as is now the case, decide upon the risks of the operation, one ought to have some more definite indications. If, for instance, the case is one of a patient whose general condition is excellent, who has no sputum and whose compressed lungs show no sign of cavity, I have not considered the indications sufficient for operation.

The unlimited possibilities of variation are, however, such that this rule cannot be absolute. If a patient has a cavity in the lung, and is without tubercle bacilli or sputum, and the lung is expanded by adhesions and is technically operable, then I consider it one for cauterization. In such a case, sputum or bacilli usually appear again sooner or later.



In some cases, as Holmboe and myself have pointed out, there is no sputum, but after every introduction of gas the patient suffers an intense, irritating cough for several days, which troubles him a great deal and prevents sleep. In these the adhesions are as a rule very narrow and sometimes they do not even show on the X-ray photograph. I have not seen such symptoms in cases with extensive adhesions. I consider cases with such symptoms as full indications for cauterization, so much the more as these adhesions for the most part are very easy to remove.

Finally, the presence of an exudate influences the indications for operation. According to my experience, which, however, is not very great, an existing pleurisy always becomes worse after a cauterization. In two cases I have seen a serious empyema set in as a nearly direct consequence of this. With a pleuritic exudate present, it is also, as already mentioned, difficult to determine the operative possibilities. The pleural surfaces are covered with fibrinous membrane and granulation tissue. The difference between lung and adhesion is not apparent: everything appears on a greyish background. In the cauterization itself one has to proceed with great caution, but it is not always so difficult as imagined. The adhesions appear very thick, but their outer layer is mostly loose granulation tissue which is easy to penetrate. During pleurisy, the net impression gained is that no operation should be performed unless there are good prospects of a decidedly favourable effect.

Other circumstances, which may influence the indications for cauterization, are the occurrence of tuberculosis in other regions, such as the larynx, the other lung and other organs of the body.

Tuberculosis of the larynx is no contra-indication. Tuberculosis in the other lung permits of cauterization only if it is necessary to continue the patient's pneumothorax. In cases of tuberculosis of other organs the same rule holds.

A summary of the indications for cauterization of adhesions will, therefore, be as follows:—

(1) Cord-like adhesions up to the thickness of a little finger, found by X-ray examination, can always be burnt off, and this operation can, as a matter of fact, be done without great risk. The clinical result depends also upon other factors of which I cannot give particulars here.

(2) Membranous adhesions are similarly suitable for cauterization. The possibility of operating upon them is best decided by thoracoscopy.

(3) With regard to surface adhesions, there is need for great precaution; and one should burn off only the granulation tissue which attaches the lung to the chest wall. By cauterizing the lung itself there is the risk of hæmorrhages, as well as opening up of tuberculous foci and cavities, with a consequent infection of the pleura.

#### CRITICAL SURVEY OF OPERATIONS SO FAR PERFORMED.

In a critical survey of the seventy-five cases, which I have already published, I will first consider those complications which followed operation immediately or ultimately. To begin with, we must refer to the cutaneous emphysema of greater or less degree which appears at the points of puncture of the chest wall. This complication may cause trouble for one or two days, after which it disappears and is of no further consequence. Of greater importance, however, are the pleuritic exudates which may follow operation. In the following table I have set down the various results which occurred in my cases:—

(1) Cases without exudate ... ..	36
(2) Cases with slight exudate ... ..	19
(3) Cases with long-lasting exudate and fever (6 exudate before the operation)...	11
(4) Cases with long-lasting exudate with empyema (4 exudate before the operation)...	7
(5) Cases with exudate first appearing one to three months after operation ...	2
Total ... ..	75

The result in the first group of cases has naturally turned out very favourably. After a few days of fever the patients have the same low temperature as before operation. The same thing can be said of Group 2, in which we have a slight exudate which does not reach above the dome of the diaphragm. In one or two weeks the exudate has disappeared completely. These pleurisies have, therefore, no influence on the clinical result, and one is entitled to say that the operation in four of five cases has had no unfavourable influence on the clinical progress. The third group comprises four cases in which the exudate together with a higher temperature and apparent influence on the general condition has continued for four to six weeks. Judged as a whole, the cases in this group had an ordinary tuberculous pleurisy.

In the fourth group, which also comprised four cases, the pleurisy developing after the operation was at first of a serious nature and thus of the same character as in Group 3. A tuberculous empyema appeared after one or several months. In these cases the complication has had such a very unfortunate effect, that of these four cases three ended in death after one or two years, death without doubt in no small degree caused by the general condition being weakened through the chronic empyema. In the last group the condition has been good after the operation, but after a few months an exudate has appeared which in both cases turned to empyema. Nevertheless, both patients became better later on, so that the prospects for the future are tolerably good. It is impossible to decide with certainty whether the cauterization had anything to do with the subsequent pleurisy. I believe that the empyema may have developed independently.

Gravesen's statistics show two cases of empyema and four of serous pleurisy out of sixteen cases. Of Holmboe's twelve cases, one had slight pleurisy and one severe, acute pleurisy and empyema with mixed infection, from which the patient died after four or five days. Taking the experience of all observers together, it would seem that pleuritic exudate and empyema are about the most serious complications of this operation. The mortality of my cases has been about 6 per cent., which is, however, a maximum figure and should hardly be attributed wholly to the operation. On the other hand, it is evident that this complication is not so grave as to contra-indicate the method in suitable cases.

I shall now pass on to the credit side of the procedure and present in a table the results of operations in my own cases. I have arranged the results in three groups, according to the position of the adhesions in the chest cavity:—

	Number of cases	Cauterization sufficient for complete collapse of lung	Clinical result good	Incomplete cauteri- zation
Jacobaens:—				
(a) Apex adhesions ...	10	9	8	1
(b) Lateral adhesions ...	62	44	41	17
(c) Diaphragm adhesions ...	3	3	1	0
Total ...	75	56	50	18
Holmboe ...	12	7	7 (?)	5
Gravesen and Saugman ...	16	9	7	7
Somme ...	11	9 (?)	7	2
Unverricht ...	7 (+2)	4 (+1)	4 (+1)	3
Total ...	121	85	75	35



To begin with, we have the apex adhesions. They are mostly short and technically difficult to reach with the galvanocautery. During cauterization pain is very often felt because of the proximity of the parietal pleura. In nine out of ten cases, the operation has succeeded technically and resulted well clinically in eight cases. The second group, lateral adhesions, comprises the main part of the cases. In forty-four of them the operation was successful technically and in all but two was also followed by a favourable clinical result. In these two an empyema with the above mentioned sequel developed.

In the third group, diaphragm adhesions, the technical difficulty is that the patient during the progress of the cauterization proper must hold his breath, because otherwise the adhesion will be in constant movement. What is advantageous in such cases is the fact that their cauterization is entirely painless. In all cases of Group 3 the technical result of operation was successful, but only one case turned out well clinically. The lung had extensive adhesions in the upper part of the chest, adhesions which it was not possible to remove by this method. In removing diaphragm adhesions the aim of the operation has been to get a better compression of the lung in the upper part of the chest cavity. This is, according to my opinion, possible only in exceptional cases.

Thus, the sum total of cases with clinically successful result has been fifty. Of the eleven cases in which only incomplete cauterization has been possible, in only one have I had a severe protracted pleurisy.

With regard to other authors, Holmboe had seven successful clinical results in twelve cases. Out of sixteen cases, Gravesen had nine technically successful, seven of which were bacilli-free. Two of the patients in the incompletely cauterized cases took a change for the worse because of empyema and protracted fever. The cause seemed to be an attempt to extend the indications for operation by burning off rather extensive adhesions. Among his cases it was not unusual for the cauterization to take place at several sittings, each of a duration of one hour or more. It is evident that the danger of exudate in such cases must be rather great.

If we now summarize the results of all these 121 cases, published thus far, we find that in eighty-five this method has been technically and completely successful in removing adhesions which prevented complete collapse of the lung. Naturally, the clinical result has not been so favourable, as only seventy-five have been symptom-free. By again referring to the first table, we see therefore that in these cases of adhesions one can better their prognosis so that it increases from 33·3 and 11·1 per cent., respectively, to not less than 70·2 per cent. The mortality index would, according to the same table, fall from 66·66 and 86·7 per cent. respectively, to 23·4 per cent. Whether this in reality was so in the cases hitherto operated on I cannot say, partly because the time which has elapsed since the operation is too short and partly because patients have been sent to different sanatoria and their further progress has not been under observation. A rapid survey of the facts available now would give a less favourable figure, since they point to a death index of between 30 and 40 per cent., which, however, of course has nothing to do with the operation itself. Many factors surely enter into play. The most common appears to have been that the patients were from the poorer classes and therefore unable to get proper nursing. The adhesion cases are often more severe than those in which a complete collapse is obtained at once.

Although it has been impossible by this method to obtain as high a percentage of clinically improved cases as in cases of simple, uncomplicated

pneumothorax without adhesions, the procedure ought to be of permanent value in perhaps a limited number of pneumothorax cases with cord-like or membranous adhesions.

*Case 30.*—G. C., accountant, born 1887. Onset of illness, September, 1918. Admitted to Romanias Sanatorium, November 20, 1918. Pneumothorax established

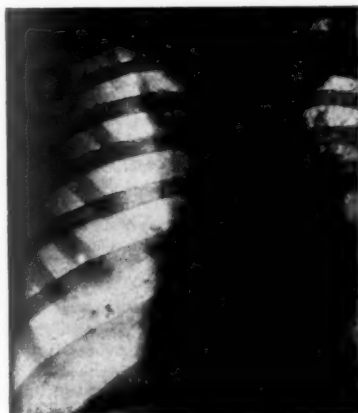


FIG. 1 (Case 30).—Apex adhesion, before the operation.



FIG. 2 (Case 30).—After the operation, complete collapse.

about March 20, 1919. During whole stay at sanatorium, temperature  $37^{\circ}$  to  $38^{\circ}$  C.: no particular improvement. Tubercle bacilli in sputum. Patient admitted May 18, 1919, for cauterization. General condition good. Sputum purulent, about 25 to 50 c.c. daily: numerous tubercle bacilli. X-ray examination, May 19, 1919: Pneumothorax on left side; upper lobe, left lung, not collapsed and large cavity at apex: extensive adhesions down to second rib impeded the collapse of the lung (fig. 1). Thoracoscopy, May.20, 1919: Lung only moderately compressed, with distinct lobuli:

only one single broad adhesion with apex at uppermost limit to be seen. For some time following, frequent refillings of nitrogen given. Operation, June 3, 1919: Lung now considerably more compressed, with the adhesion extending upward, more stretched: cauterization of offshoots of adhesion extending downward attempted: these cauterized together with overlying pleura on under side of adhesion: then attempts made with chisel to loosen bluntly adhesion from the parietal pleura: this apparently went well, but soon small hæmorrhage occurred and procedure was discontinued: moderately severe pain during cauterization, less during blunt dissection of adhesion with chisel: no reaction afterward. X-ray examination: Not different from last examination except for small notch in adhesion. Pneumothorax fillings continued with rather high pressure twice a week. X-ray examination, July 18, 1919: Condition of the adhesions at the apex of the left lung on the whole unchanged. To decide presence of cavity in lateral apex or of air spaces connected with pneumothorax cavity, which might be separated by band-like adhesions, an X-ray examination was performed in high pelvic position after injection of 300 c.c. salt solution into the pneumothorax cavity. In this position all markings at apex were found denser than before, which indicated that they communicated with the pneumothorax cavity. Second operation, July 22, 1919: Remains of previous cauterization noted as greyish-blue spots on lung surface at thoracoscopy. At present cauterization, tissue within adhesion found particularly firm and resistant. Cauterization as far back as possible and continued forward. Suddenly an empty space appeared: this was defined in front by a short adhesion. As far upward and forward as one could reach, the thin adhesion was first cauterized. The thick adhesion became narrower and also soft and limp, upward and backward. Further cauterization was therefore impossible. For a considerable distance, and, as it seemed, enough to collapse the cavity, the lung was loosened laterally. No hæmorrhage occurred. Pains, radiating to the left arm, were now and then very severe. At X-ray examination the lung was found to be loosened right up to the middle of the dome. Patient discharged August 4, 1919, on the whole feeling very well. Pneumothorax treatment at the dispensary continued. Cough subsequently increased a little. No marked and energetic compression was undertaken because of stomach trouble. The latter was evidently due, as was discovered later, to severe psychic shock. On September 8, 1919, or a month and a half after second operation, patient suffered a sudden fit of shivering with fever and slight stitch in the left side. This increased, and dyspnoea set in; and, after a few days a clear exudate was found, which revealed no bacteria on cultivation. The left half of the chest was under strong tension, and on September 14 aspiration of 900 c.c. of gas brought considerable relief. During the following days, 400 to 500 c.c. gas were aspirated every day or two. Gradually the pain diminished. Temperature, which had been as high as 40° C., fell to between 38° and 39°. However, the patient's general condition became considerably worse. Patient again admitted to Serafimerlasarett, September 25, 1919. His condition was now bad, although since the stitch appeared in the left side his sputum had ceased. X-ray examination: Lung completely collapsed; no visible cavity (fig. 2); lower third of pleural cavity filled with exudate. Aspiration of 1,300 c.c. thickened turbid opaque fluid September 29. Thoracoscopy: Pleural surfaces very red and fibrinous; here and there small hæmorrhages; lung distinctly compressed and covered with fibrin; upper lobe conical with apex adherent to dome; regions on lung surface where cauterization had been previously performed, also covered with glistening, white, fibrinous, membranous areas; no visible fistula anywhere. Later, during the autumn, typical tuberculous empyema developed with small fistule on the chest wall. In spite of this, the patient's state gradually became afebrile and by April, 1920, he had increased 20 kg. in weight. Pleural exudate had to be drawn off only at intervals of from one to two months. As far as one can judge at present, his prospects are rather good.

This case is of great interest from several aspects. Its main problem lay in the apical adhesion, which has always proved difficult of removal. This particular case was no exception to the rule. At first I tried only to loosen the pleura around the adhesions, partly bluntly with a chisel, and then

attempted to get further loosening of adhesions from the chest wall by high intra-thoracic pressure. This did not succeed, although high pressure was kept up for six weeks. The operation that followed was rather troublesome. The adhesions were fibrous and very short. The pain for that reason was very severe. Complete severance of the adhesion was not accomplished, but enough was burnt off to ensure complete compression of the lung. Before this was done 300 c.c. of salt solution were injected for diagnostic purposes and to decide whether we were dealing with a real adhesion and not the wall of a cavity. In this way it was made certain that there really was a cord-like adhesion. The immediate result of the operation was satisfactory, but in spite of this the patient was incapable of recovery on account of a very bad shock to the nervous system, about which we did not know anything at that time. Any attempt at stronger compression was not made. Six weeks later a violent acute serous pleurisy developed, which in time became converted into a tuberculous empyema but without mixed infection. It is a question whether the empyema was due to the operation. I have never been able to be clear about this. One would have thought that if the operation was the cause of it, the empyema ought to have appeared long before it did, as has happened in other observed cases. Further, this is not such a very rare complication in pneumothorax treatment in general. Yet, on the other hand, it is possible that after the operation a weakening of the lung wall opposite a cavity occurred and that the infection of the pleura took place from this. In spite of all this, the case turned out favourably, with complete collapse of the lung, cessation of the sputum and marked increase of weight. Without doubt, in spite of complicating features, the operation had a highly beneficial effect.

*Case 38.*—E. K., architect, aged 35. Pulmonary tuberculosis diagnosed December, 1918. Pneumothorax induced early in May, 1919. The general condition improved, but sputum and bacilli continued. Admitted, January 19, 1920, for cauterization. General condition good. X-ray examination: Lung broadly attached in upper part of pleural cavity down to second rib and also by coarse, band-like adhesions to middle of diaphragm, (fig. 3); medium-sized cavity in uncollapsed part of lung. Operation, January 21, 1920: Thoracoscopy shows three cord-like adhesions towards diaphragm, the anterior one of the thickness of a small finger, the middle like a thick string, and the posterior one a little coarser and continuing backward as a membrane; the middle one moved particularly with respiration; above, the lung was apparently densely attached to the chest wall, but no details could be seen. Cauterization of the middle and posterior adhesions was carried out smoothly, but the anterior adhesion was rather difficult to cauterize partly because of its fibrous nature and partly because the patient had to hold his breath every time a piece was burnt off by the cautery. The cauterization was performed without the slightest pain or hæmorrhage but took rather a long time. After the operation a small exudate developed which, however, did not reach above the middle of the dome. Test puncture revealed a clear, serous fluid with lymphocytes. This was negative for guinea-pigs. No rise of temperature above 37.5°. Sputum, which before the cauterization was 10 c.c. daily, gradually disappeared entirely. No tubercle bacilli on February 22, 1920, or afterward. X-ray examination, January 24, 1920: fluid to level of middle of dome of diaphragm; lung obviously better compressed; cavities above are smaller but still visible (fig. 4).

This case is of more than ordinary interest. It was a case in which it was desired, by cauterization of the adhesions to the diaphragm, to obtain a better compression of the upper portion of the lung, which was broadly adherent to the thoracic wall. The rapid and favourable effect of the operation (freedom from sputum and bacilli in about a fortnight) showed definitely that our course of action had been right. It is also interesting to note that this improvement

appeared before the visible cavities had quite disappeared. This indicates that sputum had come from other parts of the lung, which had been compressed by the cauterization. From a technical point of view the operation was rather difficult, because the actual cauterization had to be performed in small stages, during which the patient had to hold his breath each time.

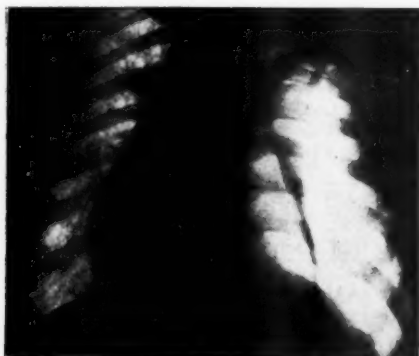


FIG. 3 (Case 38).—Adhesion to diaphragm before the operation.

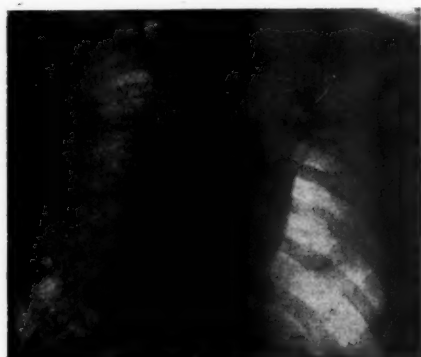


FIG. 4 (Case 38).—After the removal of the adhesion.

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Dr. S. VERE PEARSON<sup>1</sup> said that he was convinced from personal experience that a proper investigation of the nature and extent of adhesions in cases of artificial pneumothorax was an essential element in the successful treatment of these cases, and that such investigation involved in many instances inspection through the thoracoscope. The other most important means of finding out about adhesions was careful screening of the patient in the X-ray room. The patient must be in the upright position and must be revolved so that the rays passed from different angles through the body thereby often giving better indications of the thickness of adhesions and of the direction in which they traversed the cavity. He had looked into the pleural cavity in five cases so far. On the first two occasions it was at Veilefjord Sanatorium, Denmark, during the summer before last, just before and just after Professor Saugman had cauterized some adhesions. He had performed thoracoscopy himself for the first time on July 6 this year (1922). Through the kindness of his friend, Dr. Peter W. Edwards, at that time medical superintendent of the Bramcote Sanatorium, near Nuneham, he had thorascoped some of his patients there. So little did the operation upset the patients that they were in two instances polishing the floor of the wards in the morning the next day but one after the operations, which were performed late in the afternoon under careful local anæsthetization preceded by a small dose of scopolamine and morphia; and one of the patients actually went to sleep during the course of the proceedings. Unfortunately it was in this, the third case of the four he and Dr. Edwards had chosen at Bramcote, that the instrument failed. A short circuit developed in the distal part of the thoracoscope, and it was while attempts were being made to rectify it that the patient went to sleep with the cannula in his chest. At that time he had not got a second instrument at hand, and was not able to get the defective one repaired for many weeks; hence he had been unable to proceed to deal with the fourth patient. This was a great disappointment to him because this was the case he had purposely left to the last as being a patient with a thin lateral adhesion, which he had hoped to find an easy one upon whom to perform his first cauterization. He had written last week to Dr. Edwards' successor at Bramcote to inquire for the latest news of these patients, and the following information about their condition four days ago would be of interest. All were maintaining an improved state of health fairly well. The fourth case, the patient who had not been touched, was reported to have "very little cough and sputum and his general condition is fair . . . His exercise capacity equals about three miles a day walking." The question occurred, would this particular patient, whose long lateral adhesion was not interfering very seriously with the collapse of his right lung, have got into a

<sup>1</sup> Remarks made following Dr. Jacobæus' lecture at the Royal Society of Medicine, November 22, 1922.

materially better state now, three and a half months later, had that adhesion been cauterized? The third patient was now employed in the carpenter's shop and "did a full day's work without resting. His general condition was most satisfactory." Now the adhesions present in this case had deterred him (Dr. Vere Pearson) from attempting cauterization, but had he had Professor Jacobaeus' experience he would have tackled them because they seemed to be holding up the lung very definitely from a good degree of physical collapse, though, it was to be admitted, judging by clinical symptoms they were not interrupting progress.



## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

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### A New Apparatus for measuring Sensori-motor Reaction Times.

By M. D. HART, M.Sc., A. Inst. P., and W. WHATELY SMITH, M.A.

#### (I) INTRODUCTORY.

THIS apparatus was devised by the authors at the instance of Dr. C. B. Heald, C.B.E., Medical Adviser to the Director of Civil Aviation. Dr. Heald believed that sensori-motor reflexes might prove a valuable guide to the changes in the physical condition of pilots as they came for their six-monthly re-examination; but having had the opportunity of observing the appliances used for this purpose by the French authorities he formed the opinion that a greatly improved apparatus would be necessary in order to obtain trustworthy results. He therefore submitted the problem to the authors, and the present paper describes an arrangement which is believed to be superior to the methods hitherto in use. The mode of application is also made the subject of a few concluding observations.

#### (II)

Any apparatus for measuring sensori-motor reaction times must possess certain characteristics if its indications are to be of real value. These may best be illustrated by considering an example in which the stimulus is visual. The sequence of events here is as follows:—

The observer presses a key, in this way both lighting—or exposing—a lamp, and in some way determining the commencement of a time period which is terminated by the subject pressing another key as soon as possible after seeing the light. The time actually to be measured is that which elapses between the light first falling on the subject's retina and the beginning of his movement of the key.

In the course of the process, however, there are several stages at which errors may be introduced, all of which tend to make the apparent reaction time too long. Of these sources of error the chief are:—

(a) Lack of simultaneity in the beginnings of the two operations performed by the pressure of the observer's key.

(b) Lapse of significant periods during the progress of the two operations, i.e., the time required for the lamp to attain its proper luminosity or to be

fully exposed, and for the time-recording mechanism to determine the commencement of the reaction period.

(c) Lapse of a significant period between the actual end of the reaction time and the beginning of the operation performed by the pressure of the subject's key.

(d) Appreciable lag in the functioning of the mechanism determining the end of the reaction time.

In addition to these sources of error, which must be eliminated or reduced to a minimum, the following practical requirements as regards convenience of working should also be satisfied: (1) The apparatus should be capable of giving direct readings, i.e., it should not be necessary to make measurements or calculations in order to obtain the reaction times. (2) It should also, if possible, re-set itself automatically and should be strong, trustworthy, and clean to use. (3) For certain purposes the possibility of taking a number of readings in rapid succession would be an additional advantage.

Existing forms of apparatus seem to be deficient as regards some or all of these points. The ordinary electric lamp, for instance, obviously takes an appreciable time to attain full luminosity and the use of a shutter involves mechanical parts the inertia of which must inevitably introduce a certain lag. The same applies to any form of mechanical chronometer, and the reduction of lag by lightening the working parts is necessarily attended by a considerable decrease in robustness. This last point can, it is true, be met by the use of a string galvanometer and photographic recorder, but this method has many disadvantages. It is costly, complicated and unclean in use; it necessitates a certain delay for the development of the sensitized paper and involves careful measurement or counting by the observer, i.e., it does not give direct readings. The expense of renewing the sensitized paper is also worth considering and the renewal itself is tiresome. Similar considerations apply to the use of magnetically operated pens or scribes writing on charts, and in these cases also, renewals of some kind are necessary from time to time.

### (III)

In order to meet the somewhat rigorous requirements which have just been described the authors decided to use a Neon lamp as the source of light and a purely electrical method for measuring time. Neon lamps are cheap, strong and give a very constant degree of luminosity. They also possess the very valuable property of an almost complete absence of lighting lag. The exact value of this lag is not known, but as such lamps can be made to light up and go out again as rapidly as 15,000 times a second or more, it is clearly negligible for our present purpose. The Neon lamp thus abolishes one of the major sources of error mentioned above; the remainder are met by the electrical method of time measurement now to be described.

This method consists essentially in the use of a condenser the discharge of which through a resistance of suitable value is started at the beginning of the reaction time by the observer's key, which also lights the Neon lamp, and is stopped by the subject's key at the end of the reaction time. The ratio of the remaining charge on the condenser to that present at the beginning of the discharge is a measure of the reaction time. The measurement of the amount of charge is made by the use of a suitable electrometer or electrostatic voltmeter, the only essential feature of which is a high insulation resistance. If it be desired to take a series of readings in rapid succession the moving

parts and damping must be so arranged as to bring the instrument to rest within the time available.

The simplest form of the circuit is shown in fig. 1. In this figure,  $K_1$  represents the observer's key,  $K_2$  the subject's key,  $L$  the Neon lamp,  $E$  the electrometer,  $C$  the condenser,  $R$  the resistance and  $V$  the leads of ordinary 200-volt lighting mains. When  $K_1$  is in the position shown current flows through  $R$ , and  $C$  is kept charged to the potential of the mains. When  $K_1$  is depressed the circuit through the lamp is made and the connection from the mains to the condenser broken so that the latter tends to discharge through the resistance  $R$ . The moment  $K_2$  is depressed the connection between the condenser and resistance  $R$  is broken and the discharge is stopped.

It is well known that the drop of voltage across a condenser while it is discharging through a resistance is exponential with respect to time and that it falls to  $1/e = 0.3679$  of its original value in a time equal to the product of the capacity into the resistance through which it is discharging. If this product be made equal to about a second the instrument will be adapted to the measurement of reaction times. Since the time taken for the voltage across the condenser to fall from its maximum to any given value is a function of that value alone the scale of the instrument may be calibrated direct in time. The calibration is, of course, arranged so as to give a zero reading when the

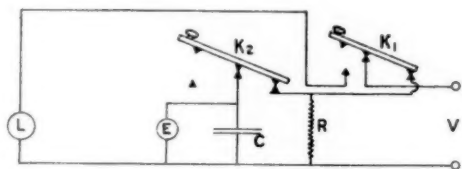


FIG. 1.

condenser is fully charged. The method of calibration is described in Section IV below.

The apparatus actually exhibited is elaborated in certain respects. It is provided with two lamps and two corresponding keys for the subject. There is also a second observer's key adapted for measurement of the subject's reaction time for auditory stimuli.

The circuit diagram is shown in fig. 2 in which  $P$  is a potentiometer arranged so that the instrument may be adjusted to zero in spite of variations of the supply voltage;  $C$ ,  $E$ , and  $R$  are respectively the condenser, the electrometer and the resistance;  $K_3$  and  $K_4$  are the subject's two keys corresponding to the lamps  $L_1$  and  $L_2$ ;  $K_1$  and  $K_2$  are the observer's "visual" and "auditory" keys.  $S_1$  is a telephonist's three-way switch, the right and left positions of which put lamps  $L_1$  and  $L_2$  respectively in circuit with the appropriate key  $K_3$  or  $K_4$  (the key not corresponding to the lamp in use being disconnected). The central position disconnects both the lamps  $L_1$  and  $L_2$  while rendering either of the keys  $K_3$  or  $K_4$  available for use at will, and it is intended to be used for auditory tests, in which case it is necessary also to switch over  $S_2$  to the "auditory" key  $K_2$  instead of to the "visual" key  $K_1$ . The two-way switch  $S_2$  is for use with the calibrating device (see Section IV below).

Terminals T T are also provided in case additional voltage, which may take the form of dry cells, is necessary if the main supply be not high enough to operate the Neon lamps.

The "visual" key is provided with spring contacts in order to minimize the sound of operation, whereas the "auditory" key has brass contacts of which the click is distinctly audible to the subject. Alternatively the "auditory" key may operate a bell, buzzer or acoustic oscillator, and may convey the stimulus to the subject through head telephones or a loud speaking telephone. Where an oscillator is used it is easy to arrange for a stimulus of variable pitch so that reaction times for different pitches may be studied.

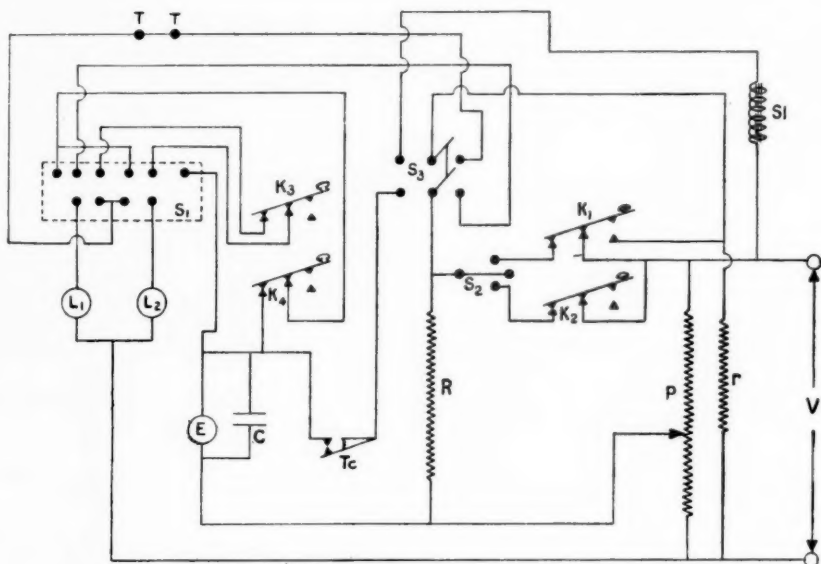


FIG. 2.

The merits of this arrangement are best brought out by considering seriatim the four possible causes of error described earlier in this paper:—

(1) The first of these was the possibility of the two processes performed by pressure of the observer's key not starting simultaneously. As regards this point the present apparatus is not theoretically perfect, since some very small time must clearly elapse between the breaking of the one contact which determines the beginning of the reaction time period and the making of the other which lights the lamp. This cause of error is however removed by the method of calibration, and the only remaining error is a second order effect arising from differences in the rate of pressure of the key, or from differences between the two keys,  $K_1$  and  $K_2$ . Moreover, in practice the clearance between the two parts of the second contact can be reduced to any desired degree of

smallness—say one-thousandth of an inch—and the error from this cause may therefore be rendered quite inappreciable, even apart from the calibration which removes it.

(2) The second point, namely, the possibility of lag in the performance of the two operations thus started is completely overcome by the use of the Neon lamp and of a purely electrical method of timing. The Neon lamp attains its full luminosity practically instantaneously, and since there are no mechanical moving parts in the timing device there is no inertia to be overcome; consequently the process of time recording starts absolutely without lag.

(3) Since the pressure of the subject's key is only required to break a circuit this must occur the very instant the pressure is applied, and this covers the third point raised.

(4) Finally, the same considerations apply to the arresting of the time-measuring process as to its initiation: there are no mechanical moving parts and the discharge of the condenser is stopped absolutely instantaneously when the circuit is broken by the observer's key.

With respect to the other desiderata enumerated it may be noted that the scale of the voltmeter can easily be calibrated directly in fractions of a second and that consequently no calculations or measurements of any kind are necessary in order to obtain the reaction times. The apparatus resets itself automatically, and is perfectly clean and easy to use; it is also simple and unlikely to get out of order.

It is true that the particular form of voltmeter used in the demonstration set is of a type which takes some little time to come to rest and consequently it is not possible to take a rapid succession of readings. But this is, of course, merely a matter of the design of the voltmeter, and although inquiry among the leading instrument makers has so far failed to discover a suitable short-period instrument at a reasonable price, there is no reason, theoretical or practical, why a simple quick-acting electrometer or voltmeter should not be produced at a low price, and in the event of a demand arising there is little doubt that it could easily be met.

The actual arrangement demonstrated—namely, that for two visual signals or one auditory signal—is, of course, quite arbitrary and has merely been selected by the authors as a representative sample of the potentialities of the method. They realize that it may not meet every requirement in this class of work, and would be glad to receive suggestions as to what is needed for particular purposes. It is, of course, easy to provide an instrument covering two or more ranges of time periods as well as to alter the number or type of stimuli used.

#### (IV)

It may also be pointed out that it is easy to fit, as a permanent accessory to the apparatus, a device which will enable it to be calibrated with accuracy as often as is desired, and such a device is embodied in the demonstration set. This has the advantage that there need be no fear of inaccuracies being introduced through variations with time such as might be caused by change in the capacity of the condenser, weakening of the insulation or change in the resistance through which the condenser discharges. The device operates as follows:—

A steel ball of suitable size is held up by an electromagnet *Sl* (fig. 2)—the coils of which are in series with the mains and a resistance *r*, the appropriate contacts of the observer's key being in parallel with the coils of the solenoid.

When the latter is depressed the current in the solenoid is diverted and the ball falls through a suitable distance on to a small trigger  $T_c$  which corresponds to the subject's key and, in precisely the same way as the latter, stops the discharge of the condenser by breaking the circuit.

Thus, if the ball be allowed to fall through a distance of 1 ft., the time elapsing between the starting and stopping of the condenser discharge will be almost exactly 0.25 second—its precise duration depending, of course, on the local value of "g," the acceleration due to gravity—which may normally be taken as 32.2 ft. per second per second without error.

By altering the height through which the steel ball drops a number of calibration readings may, of course, be obtained if desired and a complete calibration curve may be plotted: this would be essential for the primary calibration, but unnecessary for occasional check calibrations.

It should be noted that the calibration circuit is so arranged that the current through the solenoid does not fall until the instant at which the circuit is made through the third contact of the observer's key. That is to say the calibration time period does not commence until the corresponding instant at which the time period to be measured commences in an actual test; hence the error due to the non-simultaneity of the commencement of the time period and the operation of the time-recording mechanism is counteracted.

#### (V)

We conclude this paper with a few observations on the use of the apparatus and the information which it seems reasonable to suppose may be derived from the reaction times which it measures.

There are three main qualities of reaction times which may be recorded and which might, on *a priori* grounds, yield information of value. The first is the absolute mean magnitude of any given series of times; the second is their variability within that series; the third is the alteration in their absolute magnitude or in their variability from one occasion to another.

It seems to be generally conceded that the first of these is of little value for practical purposes, at any rate where the testing of air pilots is concerned. It is, of course, obvious that a pilot who is able to react quickly to a situation is to a certain extent superior to one who reacts more slowly. But we do not know whether this power of quick reaction is one of major importance or whether a 10 per cent. variation in it represents an equivalent variation in the pilot's ability. On the whole it seems likely that it does not. If the tests show that a particular pilot can in general react to a situation in, say, one-tenth of a second, it does not seem to matter very much if another pilot requires as much as one-ninth to perform the same reaction. Moreover, we have no good reason for supposing that there is any direct connection between the reaction times for the artificial situations which we produce and those which actually occur in practical flying. All we can reasonably claim is that in each case the nervous system is implicated and that if the response to the real situation be one function of this, the response to the artificial situation is another: both functions presumably involve the contemporary state of the nervous system, and, therefore, the reaction times to the two kinds of situation are likely to show a positive correlation. That is to say, if one be unduly prolonged the other in the same individual is also likely to be so; if one be very variable so may the other be expected to be—and so forth. But we cannot reasonably expect to be able to assess the absolute magnitude of the one by measuring the absolute magnitude of the other.



If we measure the mean reaction times of a considerable number of pilots and find that they vary between—let us say—one-tenth and one-fifteenth of a second, and some other pilot gives us a mean time of, say, one-fifth of a second, we can, it is true, conclude that the latter is appreciably different from the previous pilots; but it is doubtful whether we are entitled to conclude that he is likely to be on the whole a worse pilot; it is even possible that he may be better; he may take longer to react but be able to think more effectively about what reaction is best.

On the other hand it does seem likely that a series of times the variability of which is very large represents a less settled or reliable state of the nervous system than one the variability of which is small. A man who can react time after time with perfect regularity is almost certain to have his nervous system in good condition, whereas a jerky and irregular series of times ranging from very short to very long times is clearly suggestive of a nervous condition requiring attention.

Even more important, probably, is the difference between the reaction times given by the same pilot on different occasions. If a man at several successive examinations gives a mean time of, say, one-tenth of a second and never deviates from this mean by more than 10 per cent., we may reasonably regard this as his standard reaction time when in normal health. If on some subsequent occasion he suddenly gives a mean of, say, one-fifth of a second it is fairly clear that in some respects, at least, he is abnormal, and it would, therefore, be wise to recommend him not to fly for a while. The same would apply if his mean reaction time suddenly decreased to one-tenth of a second.

This appears to be a trustworthy and genuinely valuable test and one which on account of its simplicity and speed is very well worth applying to professional pilots. Inasmuch as the elimination of those causes of error which tend to increase the reaction time will necessarily make the results of tests more truly comparable the authors venture to hope that the apparatus here described may prove of value to medical science and to practical flying.

Squadron-Leader T. S. RIPPON said that from his experience of reflex reaction time tests on aviators, he quite concurred in the statement that the important thing was the degree of variability in the reading, which was the indication of nervous stability. In the Royal Air Force they had carried out a considerable number of tests with D'Arsonval's chronoscope and the pendulum chronograph. They used, in addition to simple visual and auditory stimuli, the dilemma between two different coloured lights, and found that this accentuated the difference between the nervously stable and unstable. At the present time they did not use reflex reaction time tests as a routine method in assessing candidates, but only for special cases, because it had been found that the history given by the candidate of his aptitude for games at school, which was subsequently checked when he was admitted, had given them the necessary information as to the rapidity of his reflexes. Should the candidate develop any nervous instability during his flying career, they used the D'Arsonval chronoscope to determine the extent of the variation of the responses. During 1918 an interesting series of tests had been carried out on pilots and pupils under instruction in flying at Northolt aerodrome by the late Dr. Stamm, who fitted up a complicated dilemma test apparatus with mixed visual and auditory stimuli. The effect of the different lights, combined with electric bells, introduced another factor into the test, namely, an emotional reaction, and it was seen that the best types of flying men were unemotional, and not easily flurried by the rapidly changing stimuli. On one occasion he (the speaker) had had the opportunity of seeing Dr. Nepper (who in conjunction with Dr. Camus had been mainly responsible for the testing of the psychomotor responses of French pilots) carry out some examinations in Paris. Dr. Nepper also used D'Arsonval's chronoscope, and when he tested the "Aces," as their most distinguished fighting pilots were called, they showed quick and even



responses, whilst pilots who had crashed, and had not recovered fully from the effects of concussion or emotional shock, showed marked variations in proportion to the extent of the nervous instability. He had also seen Dr. Nepper test a pilot in the decompression chamber, in which a pilot was examined at a pressure equivalent to an altitude of 24,000 ft. The diminution of oxygen resulted in the reflex reaction time responses being slowed to approximately double those taken at sea level. He noted that the apparatus which had been demonstrated that evening was suitable for testing both simple psychomotor reactions and also, by using mixed auditory and visual stimuli, for testing to a certain extent the emotional stability of the subject. The only criticism of the apparatus that he would make was that the voltmeter appeared to take some appreciable time before coming to rest, but he understood from the demonstrator that this would be rectified in the finished apparatus.

## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

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### Artificial Pneumopericardium.

By R. W. A. SALMOND, O.B.E., M.D., Ch.M.

IT is probably safe to say that most of the more recent advances in radiological diagnosis have been made by the artificial introduction of air or other gas into various parts of the body. The following case illustrates the value of air injection into what is probably an unexploited region, namely, the pericardium. It is frankly acknowledged that this injection was made accidentally, and that it was only after it had been done that its possibilities as regards diagnosis and perhaps also, to a much less extent, treatment, were seen. The history of the case is briefly as follows:—

The patient was 21 years of age, and an electrician by trade. On June 1 he became conscious of dyspnoea followed by pericardial pain. Three weeks later, while running to catch an omnibus, he felt a sharp pain in his chest and nearly fainted. He presented himself at hospital, and on examination showed dyspnoea, but no cyanosis, slight oedema of the ankles and a large effusion in the pericardium. He was admitted to hospital under the care of Professor T. R. Elliott.

A week later his pericardium was tapped, and about 30 oz. of brownish-red fluid withdrawn. A month afterwards the fluid had re-accumulated and he was again tapped, 50 oz. of fluid being removed. The character of the fluid was much as before, except that it contained some fresh red blood cells. On August 6 he was sent to the radiological department for examination. This showed a much enlarged cardiac shadow, with no definite pulsation, and was obviously a condition of hydropericardium (fig. 1). In the oblique view nothing could be made out in the posterior mediastinum on account of the enlarged pericardial shadow extending backwards. A week later another paracentesis was performed, and 40 oz. of amber-coloured fluid removed from the pericardial sac. Soon after this this patient discharged himself, as he felt so much better and was able to get about.

Two months later, in October, he was re-admitted, obviously much worse, more dyspnoeic and with a larger amount of fluid in the pericardium, but with no evidence of venous congestion. Another X-ray examination showed a further enlargement of the pericardiac shadow (fig. 2). A month later Professor Elliott did a paracentesis to the right of the sternum and removed 70 oz. of amber-coloured fluid. There was no distress during the puncture, but during the afternoon he showed signs of collapse, cyanosis, feeble pulse,

and vomiting. On the following day a right-sided pneumothorax was diagnosed by Dr. J. W. McNee. Two days later the patient was quite comfortable, there were no signs of pneumothorax, but Professor Elliott found definite physical signs of hydropneumopericardium, and this was confirmed by X-ray examination (fig. 3). This showed a free fluid level in the pericardium with air above, and what was more important, a defined mass to the left of midline. This was the first time that it had been possible to demonstrate with any certainty the tumour or cyst, as it eventually proved to be.

Ten days later, another X-ray examination showed a further point of interest, namely, that the fluid in the pericardium was now becoming absorbed, and at about the same rate as the air (fig. 4). A week later a further



FIG. 1.



FIG. 2.

examination showed the pericardium to be much more normal in size, and the definite mass above and to its left showed slight pulsation (fig. 5). The pericardium ultimately became normal as regards fluid, while progressive bulging, with communicated cardiac pulsation, over the first, second, and third intercostal spaces appeared as the result of the enlargement of the cyst.

The conclusion now having been reached that this must be some kind of tumour, either in or close to the upper left part of the pericardium, it was decided that an operation should be done. The patient readily agreed, and it was performed one week later by Professor G. E. Gask, of St. Bartholomew's Hospital, with most successful results. The sternum was split, but neither the pleural nor pericardial sac was opened. A large cyst, containing 40 oz.

of greenish-yellow fluid, was found closely adherent to the upper left aspect of the pericardium. It had been leaking into the pericardium and had been the cause of the fluid there. As much cyst wall as possible was removed and a drainage tube inserted. Microscopically, the cyst wall was thick, vascular, and contained cartilage, plain muscle, mucous glands, and was lined with ciliated epithelium. The patient made a splendid recovery, and six weeks later was able to walk up to hospital and have another X-ray record made (fig. 6). This shows a practically normal chest. Two and half years have now elapsed since the operation and he is in very good health and able to cycle and do his ordinary work.

One of the many points about this case is how the air got into the pericardial sac. Professor Elliott assures me that none got in by way of the



FIG. 3.



FIG. 4.

syringe when paracentesis of the pericardium was done. The most likely explanation then is that the needle on its way to the pericardium passed through collapsed and compressed lung. As the distended pericardial sac diminished in size from the withdrawal of its fluid, the lung was enabled to expand and so produce the pneumothorax which was found the day after the puncture. Later the air in the pleural cavity gradually filtered into the pericardium through the needle puncture, no doubt helped by the systole and diastole of the heart.

Another point was the effect of the air inside the pericardium. It at once started the absorption of the fluid in the pericardium, and the leakage from the cyst ceased. It is a question of medical interest whether, in the more usual

cases of pericardial effusion, the introduction of air would start or help to start a similar absorption. This has been tried in cases of pleural effusion after some of the fluid has been removed. In these cases the air causes the lung to collapse and so puts it at rest, but in the case of the heart this is naturally a much more delicate proposition.

The case described is no doubt an exceptional one, but it is felt that in certain suitable cases the artificial introduction of air or other gas into the pericardium would be of much diagnostic value. The present case



FIG. 5.



FIG. 6.

would almost certainly have terminated fatally if the cyst had not been revealed by X-rays and followed by so successful an operative result.

In conclusion, I wish to gratefully acknowledge the generous help of Professor Elliott and his assistants for the medical notes, and to Professor Gask for the surgical notes of the case.

*Note.*—The blocks illustrating Dr. Salmond's communication, and Professor George's Mackenzie Davidson Memorial Lecture following, have been kindly lent by the Editorial Board of the Archives of Radiology and Electro-Therapy.

PROFESSOR M. PHILIPPSON (Brussels) read a paper on "High Frequency Currents applied to the Study of Cellular Physiology."

[February 16, 1923.]

**The Pathological Gall-bladder :**THE MACKENZIE DAVIDSON MEMORIAL LECTURE.<sup>1</sup>

By Professor ARIAL W. GEORGE, M.D.

*(Assistant Professor of Roentgenology, Tuft's College, Boston, Mass.)*

It was with distinct pleasure that I accepted your invitation to come to England to talk to you on this most interesting subject. And further, the knowledge that this meeting commemorates the memory of one who sacrificed his life in the advancement of our specialty makes me feel humbly grateful for the honour you have extended to me and America. I refer to Sir James Mackenzie Davidson, whose work was so well known in my country and throughout the world.

## THE PATHOLOGICAL GALL-BLADDER.

No one can approach the gall-bladder problem without being familiar with the work of Robert Knox. To Dr. Leonard and myself, his publication on the "Radiography of the Gall-bladder" was probably the greatest stimulus we had in our early work upon gall-bladder disease and gall-stone diagnosis. Possibly we were a little discouraged after a careful analysis of Knox's work because of the fact that he showed there existed a large group of gall-stones which, with existing technique, could not be demonstrated by X-rays owing largely to the physical properties of those gall-stones.

In the early days of our gastro-intestinal work, when studying the duodenum by the plate method so ably advocated by Cole of New York, we occasionally found shadows either distinctly due to gall-stones or very suggestive of such a condition. We spent a great deal of time, study and effort to determine, in every case which came to our notice in our clinic, whether gall-stones were present, and, if they were found, to distinguish between those which were readily recognized and those which were more difficult to distinguish. After achieving considerably more success than we had ventured to hope for in the beginning, we found that there were many cases showing indefinite shadows, suggestive, so far as size, position and shape went, yet hardly diagnostic of gall-stones. In addition to those fairly indefinite points there was a certain amount of increase in the density, sufficiently marked to raise the question whether those shadows might not be due to a collection of gall-stones in the gall-bladder. After a period of time, during which we met with discouragement from many quarters, and especially from a number of our colleagues, it was gradually forced upon us in a good many cases in which we thought, because of the density and the shape of the shadows, that we were dealing with gall-stones, whereas, on operation, the gall-bladder was found not to contain stones, that the shadows shown might be due to the gall-bladder itself, and that the organ was rendered visible by changes in the gall-bladder or its contents, viz., chronic thickening of the walls, inspissated bile or very thick and dark bile. We accordingly changed our opinion on the

<sup>1</sup> Delivered at the First Annual Joint Meeting of the Röntgen Society and the Section of Electro-Therapeutics of the Royal Society of Medicine, May 17, 1923.

question of gall-stone diagnosis, and in our reports on cases examined altered our description and referred to the condition as "The Pathological Gall-bladder." Our interest became centred on this aspect of our work, and it became obvious from previous experience in gastro-intestinal work, and more especially from observations on the right upper quadrant of the abdomen, that we had not been able up to that period to demonstrate the normal gall-bladder; and we believe that the statement holds good to-day. There may be an occasional exception to this rule, when the normal healthy gall-bladder is shown, but speaking generally it may be safely asserted that, as a rule, in the great majority of cases we are unable to demonstrate the normal healthy gall-bladder. With this conviction in our minds we began to recognize a shadow in the right upper quadrant, consistent in size, shape and position with what we supposed the gall-bladder itself rendered visible on the negative. We concluded that what was shown was the gall-bladder, and that its visualization was rendered possible by some change within itself. This we regarded as the pathological gall-bladder with or without gall-stones. Again, we found that our colleagues did not take kindly to this observation, their efforts, aided by most of the roentgenologists, were directed to prove that the normal gall-bladder could be demonstrated, and, therefore, that our deductions were erroneous. For a time, in the work of our clinic, we stood alone in the field of pathological gall-bladder diagnosis; however, within the last few months there has been a change of opinion, and others are beginning to share our views on the subject. This rapid change in opinion was particularly brought about by one of the younger radiologists, Kirkland, of Muncie, Indiana, who was associated with me in the army and became interested in this branch of our specialty. On his return to civil practice he was fortunately located in a State and in a part of our country in which gall-stones and biliary disease appeared to be comparatively common in people of an age rendering them susceptible to those conditions. Within a year or two he made a collection almost equal to ours in number. He carried on the work on lines similar to our own, and, studying the cases from the same point of view, he obtained comparable results from the X-ray point of view. He was fortunate enough to have the surgical findings in agreement with his X-ray diagnosis in a large percentage of the cases examined. As an outcome of this he was invited to the Mayo Clinic for two months, and during that time he examined by X-rays the gall-bladder cases investigated at the clinic, diagnosed clinically as gall-stone or gall-bladder cases and prepared for operation. I have not his exact figures, but I believe that in 92 per cent. of the cases he examined the diagnosis of gall-stones or gall-bladder disease was found to be correct. The total number examined was over 100. This appears to me to be a very startling but satisfactory demonstration of the ability of a trained observer, following a comprehensive technique to prove the value of the method. I cannot personally speak too highly of my appreciation of Dr. Kirkland's work.

Later, as we perfected the technique of the examination of the upper right quadrant, there came a time when we needed, in a large number of cases, other evidence to help substantiate the visible gall-bladder, so we began to enumerate and classify observations which we called "indirect evidence" or "secondary evidence." We prefer the latter term.

#### PRESSURE DEFORMITY.

We found that the normal healthy gall-bladder did not cause pressure so that it would outline itself upon the barium mass. We also found, however, that when we obtained this pressure deformity of a certain character it was



almost conclusively due to the gall-bladder, and in almost every instance to disease of the gall-bladder, in other words, we were dealing with a pathological gall-bladder.

We classify this filling defect, or pressure deformity, in various ways. The effect of the gall-bladder pressure is exercised upon the first portion of the duodenum, in both the antero-posterior and lateral diameters. We may find in the gastro-intestinal examinations deformity of the first portion of the duodenum due to various causes, such as poor filling of the cap at the moment of exposure, malposition of the stomach for various reasons, either normal or abnormal, spasm at the pylorus, adhesions, and ulcer. I believe that most of us can recognize the characteristic changes of ulcer as easily as cancer of the stomach. There may be unusual deformities, sometimes hard to differentiate from ulcer, but we have our method of determining these with more or less certainty.

In the deformity due to a gall-bladder we have a definite and tangible shadow, which we have termed a "half-shadow," due to the convex or round surface of the gall-bladder pressing against the corresponding surface of the duodenum. To make this important point clear, we assume that the pathological gall-bladder possesses a positive pressure from the contents, either gall-stones or altered bile. The distended gall-bladder becomes spherical, and any part of its contour will be more or less convex. If the pressure is sufficiently great it will make an impression on the corresponding surface of the duodenum and when the opaque material fills the "cap" it is unable to overcome the pressure exercised by the gall-bladder, and a concave depression will appear at the part in apposition with the gall-bladder. If the edge of the liver, which is a flat surface as a rule, presses against the duodenum, we lack the "half-shadow," as we have no concavity to produce this half-shadow. My own colleagues in America have not seemed to grasp the fundamentals of pressure deformity due to the gall-bladder—i.e., the interpretation or differentiation of a pressure deformity due to a gall-bladder in contradistinction to that of a liver edge *must* be understood and recognized, to make what I am going to say clear and easy of interpretation.

#### CAUSATION OF PRESSURE.

Why should the gall-bladder cause this pressure? In the first place, we have pathological gall-bladders with no adhesions, and a great many with adhesions between the gall-bladder and the duodenum. The chances are that at some period of the examination or examinations there will be tension within a pathological gall-bladder greater than the tension within the duodenum when filled with barium. We conclude that when the stomach is in either the prone or lateral position it cannot, in view of the power exerted by gravity, peristalsis, and the weight of the barium mass, push to one side a gall-bladder in close approximation to the duodenum; we then have the feeling that tension is greater in the gall-bladder than in the duodenum. We admit that occasionally one finds a perfectly healthy gall-bladder which for a short period of time is under tension, but which empties itself sooner or later. On the other hand, a gall-bladder under tension with power to impose itself upon the first portion of the duodenum is potentially pathological in some degree.

Unfortunately, we must admit that under ordinary circumstances fluoroscopy does not help to determine these facts; their determination can only be obtained with the most careful plate or film method. Many cases show deformity not

in the prone position, but in the lateral position only, so that all views must be taken. Many cases show the pressure deformity of the gall-bladder upon the first portion of the duodenum in the lateral position but they more frequently show the picking up of the second portion of the duodenum and its extension back toward the spine in the horizontal position. It is therefore important, when one suspects a deformity due to a distended pathological gall-bladder, to endeavour in every way possible to demonstrate the half-shadow effect, because this, in our opinion, is peculiar to the deformity caused by the convex body of the gall-bladder. With gall-stones of the type invisible to X-rays we occasionally see a serrated edge to the shadow produced by the gall-bladder against the duodenum.

I have no statistics as to the relative frequency of pressure deformity and pathological gall-bladder, but to-day Dr. Leonard and I feel more convinced than ever that this deformity is one of our strongest links in the chain of evidence in the direction of the diagnosis of a pathological gall-bladder.

#### INVOLVEMENT OF SECOND PORTION OF THE DUODENUM.

The second important structure involved—although perhaps not so frequently as the first portion of the duodenum—is the second portion. Those who have carried out one method of procedure, covering a large series of cases and over a long period of time, realize the appearance of the average normal stomach. In the prone position we find that the second portion of the duodenum extends downward parallel to the vertebræ, until it reaches its third portion, where it passes upwards and behind the stomach; the position of the second portion has a great significance in the diagnosis not only of gall-bladder disease but of other conditions.

Exceptions are met with in very stout or very poorly nourished patients, or in those with scoliosis or some skeletal defect which gives rise to change in the organs generally; apart from these the position of the second portion of the duodenum is constant, as above outlined; a very common change due to gall-bladder disease is the picking up of the second portion of the duodenum outward toward the liver, and in quite a number of cases the size of the gall-bladder can be outlined by the position of the second portion of the duodenum. In the study of the pancreas, changes in the second portion form our most important diagnostic sign. In the lateral position, instead of the second portion of the duodenum passing downward and parallel to the vertebræ, it often takes a backward curve, depending upon the size of the head of the pancreas. With a nodular pancreas this pressure deformity of the nodules will be seen on the barium-filled second portion of the duodenum so clearly that there is no great difficulty in the interpretation. Again, in the lateral position, a duodenum that passes backward towards the vertebræ, and then forms an angle passing downward again to its normal position, is most significant of gall-bladder disease. Burnham, of San Francisco, at a meeting of the American Roentgen Ray Society, in September, 1922, very definitely brought out this observation in a large series of cases.

In dealing with retroperitoneal tumours—for example, sarcoma—the duodenum will be found to be pushed in the opposite direction, and the tumour mass will be outlined upon the posterior portion of the duodenum; it can be detected by the new position which the second portion of the duodenum takes as compared with the normal.

It is of course unnecessary to say that the best results will be obtained by the plate or film method; with sufficient experience this observation can be

detected fluoroscopically. The post-operative case is an exception to general rules; all the above observations may be valueless because of fixation, omental and otherwise. All cases that have undergone previous abdominal operation, especially of the upper right quadrant, are difficult to interpret, as normal relations are so definitely disturbed in some instances. We cannot urge too strongly a careful consideration of all these facts relative to gall-bladder disease. The second portion of the duodenum plays its most important part in the diagnosis of the gall-bladder region.

When the duodenum passes outward towards the liver, and then proceeds downward to its normal position, we must consider that this position of the duodenum is due definitely to *fixation*, and in only rare exceptions will this be found to be due to other than gall-bladder adhesions. When this occurs it is a very definite observation.

#### PRESSURE UPON THE ANTRUM.

Pressure of the gall-bladder upon the antrum of the stomach will occur as frequently as pressure deformity on the first portion of the duodenum; in a large number of cases it will be found on the first portion of the duodenum, extending on to the antrum. In this observation we must look for this half-shadow effect. Another observation of value is the transposition of the jejunum from its normal position from the left to the upper right quadrant. This has frequently been of assistance to us in the determination of gall-bladder disease; but we appreciate from experience that tuberculous or other peritonitis earlier in life may be the cause. It is remarkable how easily the gall-bladder throws out adhesions and picks up adjacent structures.

#### FILLING OF THE AMPULLA OF VATER.

A fourth observation of relatively common occurrence, but possibly not of quite such definite value, is the filling of the ampulla of Vater. We have found, in every instance in which we have had the cases operated upon, that if we could show the ampulla full of barium throughout a series of gastro-intestinal plates or films, this was significant of one of two conditions—gall-bladder disease or disease of the pancreas. We know, of course, changes frequently occur in the pancreas secondary to chronic gall-bladder disease. At one time we kept a record of a series of these visible ampulla cases operated upon for gall-bladder disease and other conditions to the number of twenty-seven; in every instance they displayed disease either of the gall-bladder or of the pancreas, or both. If all the other indications outlined are absent or cannot be demonstrated, and if we find only the filling of the ampulla of Vater, we feel that we have neglected for one reason or another to obtain the most satisfactory results from this method throughout the examination.

#### CHANGE IN POSITION OF PROXIMAL PORTION OF TRANSVERSE COLON.

Another observation that can be mentioned, and which is of considerable importance, is the change in position of the proximal portion of the transverse colon. In a way it forms a secondary hepatic flexure, or, as we have sometimes called it, a "pseudo-hepatic flexure." Very often, in the absence of all other signs, this change of the transverse colon will be present and will constitute the *only diagnostic* sign pointing to the gall-bladder.



FIG. 1.—Gall-stones.

One other diagnostic factor, brought forward by Carman, Burnham, and others, which we have felt was not entirely reliable, is so-called spasm which occurs in the antrum of the stomach. Possibly this should be observed with the fluoroscope rather than with plates or films; we do not attach any considerable significance to it, unless it appears in conjunction with other signs which make the diagnosis positive.

It can be readily seen that if these observations which I have enumerated are reliable, we have considerable evidence to help towards the diagnosis of a pathological gall-bladder or gall-stones, apart from the visible gall-bladder. To



FIG. 2.—Gall-stones.

me this makes the work still more satisfactory, because we are not depending upon one diagnostic observation, but upon numerous observations, any one of which may be of the utmost importance in a given case.

#### NEGATIVE VALUE OF X-RAYS.

Many have asked the question, "What is the negative value of X-rays?" We feel, perhaps now more strongly than ever, that if we have evidence which makes for a *positive diagnosis*, then the absence of all of this evidence has some value towards the negative aspect of the study. We are sure that, with a

proper examination, a given case of actual pathological processes of the gall-bladder can be recognized in almost every instance by changes manifested in the plate or film of the X-ray examination. There are, unquestionably, degrees of pathological processes, such as mild cholecystitis, which do not change the quality of the bile to any extent or give rise to thickening of the bladder wall. I do believe that *even* in these cases we can, with careful films, bring out secondary evidence of diagnostic importance. Even with cholesterin stones, which are usually in numbers, if we have a large number we have mass, and if we have mass we have increased density. It may not always be possible



FIG. 3.—Gall-stones.

to recognize individual stones, but with increased density and the shape of the gall-bladder we can be pretty sure that we are dealing with a chronic gall-bladder, or with stones, or both.

The question is then asked as follows: "Why, when we have simply the microscopic change in the gall-bladder wall, can we detect these changes with X-rays?" So far as our experience is concerned, and so far as we can find from the literature, the moment infection takes place in the gall-bladder there is change in the tissue of the gall-bladder, and immediately, or very soon, the bile becomes turbid, cloudy, considerably darker, and more tar-like. We found

in a series of laboratory tests that this bile, as it darkens in colour from the normal, became denser in quality, with increase of opacity. We have had gall-bladders removed, and with an immediate specific gravity test of the bile we



FIG. 4.—Pressure deformity of the duodenal bulb.

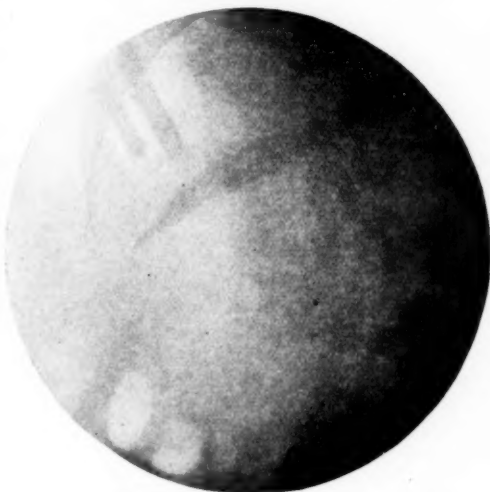


FIG. 5.—Group of small gall-stones, very indefinite.

found it was double the normal. It is perfectly obvious that with this increase in density, or specific gravity, this condition is more easily recognizable with X-rays.



## PERSEVERANCE NECESSARY.

We have been asked many times by our colleagues, especially of the younger group, why we can show things so constantly when they are unable



FIG. 6.—Visible gall-bladder, without stones.



FIG. 7.—Gall-stones.

to do so with the same apparatus and with the same type of people. Perhaps that question can be answered by stating that one big factor in gall-bladder work, aside from the necessary apparatus and facilities, is perseverance. The

investigator should continue his work in an individual case until he has obtained, through a series of films, a most satisfactory negative from every point of view. This means that the patient must be absolutely at rest without



FIG. 8.—Constant pressure of pathological gall-bladder on second portion of duodenum.



FIG. 9.—Pressure of pathological gall-bladder on second portion of duodenum.

breathing. The technique, the quality, and the exposure value must be exact for the individual case; for this there is no standard. The second big factor in this study seems to me to be a willingness to look for and to admit that these changes exist and can be found.

I think that, in view of our experience, covering a number of years and a large series of cases, we have in the use of X-rays a very definite method of



FIG. 10.—Fixation of second portion of duodenum due to pathological gall-bladder.



FIG. 11.—Constant pressure of pathological gall-bladder on first portion of duodenum.

examination of the upper right quadrant. If we do not reach 100 per cent. of correct diagnoses this does not relegate the method to a second place, for

our percentages are as high as can be expected in any branch of medical science. Up to the present time no one method touches perfection, but our



FIG. 12.—Constant pressure on first portion of duodenum and fixation of second portion of duodenum, due to pathological gall-bladder.

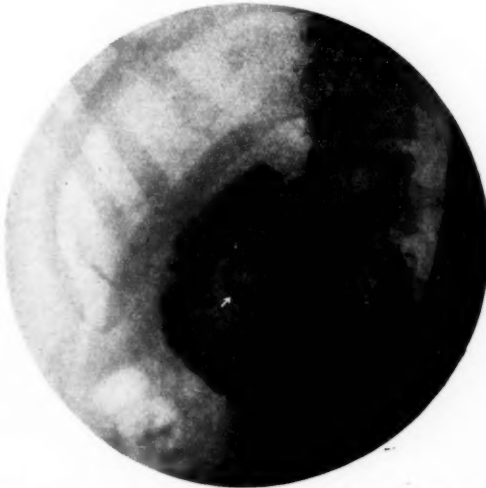


FIG. 13.—Pressure of gall-bladder on first portion of duodenum.

endeavour should be to make our practice in this speciality as *near* 100 per cent. as is humanly possible.



FIG. 14.—Visible pathological gall-bladder. Secondary pressure on first and second portions of duodenum and antrum of stomach (lateral view).



FIG. 15.—Pressure of pathological gall-bladder containing stones on antrum of stomach.



FIG. 16.—Multiple small gall-stones.

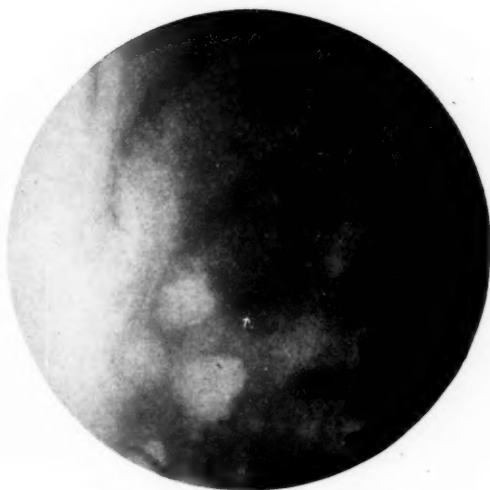


FIG. 17.—Large gall-bladder with stones.

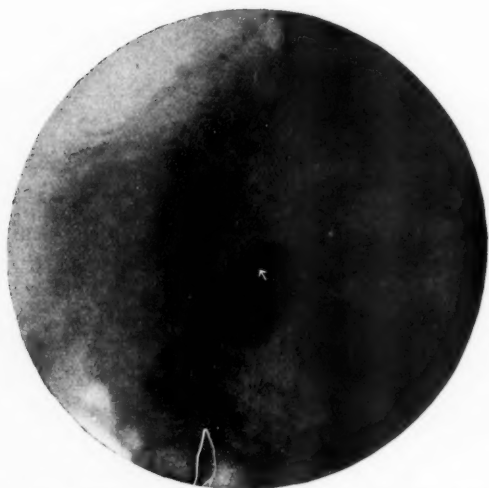


FIG. 18.—One stone and pressure of gall-bladder on first portion of duodenum.

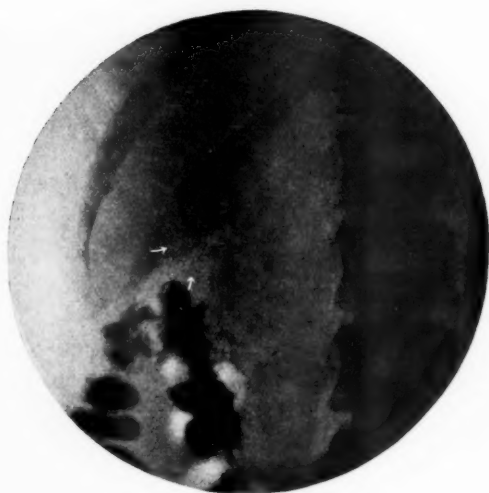


FIG. 19.—Gall-stones.



## Section of Electro-Therapeutics.

President—Dr. ROBERT KNOX.

### The Movements of the Gastro-intestinal Mucosa.<sup>1</sup>

By Professor GÖSTA FORSELL

(Stockholm).

(ABSTRACT.)

THE folds of the mucous membrane of the stomach were generally supposed to be caused by passive folding of the mucosa as a consequence of the contractions of the muscular coat. The origin of the folds of the mucous membrane of the intestine was explained in the same manner, except that here the folds of the gastro-intestinal mucosa were considered to be permanent anatomical structures ("Dauer-falten" of German authors). This general view was incorrect, and, in his (Professor Forssell's) opinion, the folds of the gastro-intestinal mucosa were formed by active movements of the mucous membrane itself. By means of several series of radiograms of the stomach, of the duodenum, and of different parts of the small and large intestine after an opaque meal or enema, he demonstrated that the same part of the stomach or intestine, although retaining the same external diameter, was able to vary the number, position, and pattern of its folds. By direct observations and serial photographs (shown at the meeting) of the exposed mucosa of the ileum and colon in patients suffering from intestinal fistulæ great fluctuations of the relief of the mucosa independent of contraction of the muscular coat were demonstrated. Anatomical preparations of the human digestive tract, hardened by the injection of formalin shortly after death, showed patterns of the folds of the mucosa similar to those seen in the radiograms of the living subject. Even the duodenal bulb, which was generally described as being devoid of folds, showed a highly varying relief of the mucosa in both radiograms and anatomical preparations.

#### MECHANISM OF THE MOVEMENTS OF THE MUCOSA.

Professor Forssell concluded from these observations that while the muscular coat (muscularis propria) of the intestine produced the well-known changes of width and external form of the digestive tube, it had no influence upon the localized and individual folding of the mucosa which he had demonstrated. The muscularis mucosæ formed the special contractile organ of the mucous membrane; being attached to the mucosa and the submucosa it was able to displace the mucosa in any direction by means of its transverse, longitudinal, and oblique fibres. The mass of the mucous membrane, and, consequently the volume or size of its folds, was regulated by the variation in filling of its vessels, while the pattern—i.e., the number, position, and form—of the folds was determined by the muscularis mucosæ. While the formation of a high and close relief of the mucosa would obviously be aided by simultaneous contraction of the muscular coat proper, a definite degree of

<sup>1</sup> The blocks illustrating Professor Forssell's paper have been kindly lent by the proprietors of the *Lancet*.

[March 16, 1923.]

contraction of the muscular coat was not necessarily associated with a definite raising of mucous folds; on the contrary, a certain degree of contraction of the muscular coat producing a certain width of the tube might be associated with a state of the mucosa varying from a total absence of folds to folding in high relief and complicated pattern.

#### RÔLE OF THE MOVEMENTS OF THE MUCOSA IN DIGESTION.

According to the present general opinion that the folds of the mucosa were anatomical and stationary, the function ascribed to them was the purely passive one of enlarging the absorbing surface of the gut and of preventing a too rapid flow of the intestinal contents. He (the speaker) held that while movements of the muscular coat determined the rough division and large displacements of the contents of the intestinal canal, it was the movement of the mucous membrane which carried on the extremely differentiated distri-

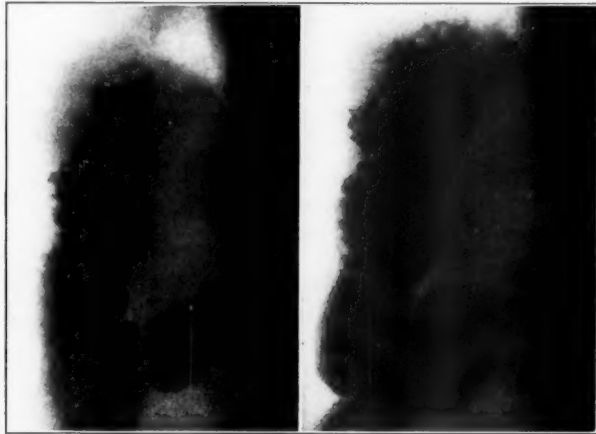


FIG. 1.—Two radiograms of a normal stomach, taken at an interval of one minute in an unchanged prone position. Compare the contours of the greater curvature in its upper half, where the mucous membrane shows deep folds; it will be observed how the folds of the mucous membrane have changed their number as well as their position and form, without any change in the width of the stomach.

bution of the food in digestive chambers of varying form and size, and controlled the fine regulation of the rate of flow along the alimentary canal. The knowledge that the folds of mucous membrane were not passive structures, but represented phases of active movement, must involve a new appreciation of their function. The fact that the whole rigid world of folds and furrows thus became alive and governed by independent motor forces might prove to be of great importance in our conception of digestion from both the mechanical and chemical standpoints. The rôle that disturbance of the motor mechanism of the mucosa might be playing in the pathology of the alimentary tract was as yet very imperfectly understood, and many difficulties might occur in working out this problem. The pathological folding of the mucous membrane was not

a subject for his comments except to say that the importance of realizing the mucous relief of the intestine was illustrated by the fact that the deformities of the duodenal bulb such as the so-called "defect" and the "retraction" of

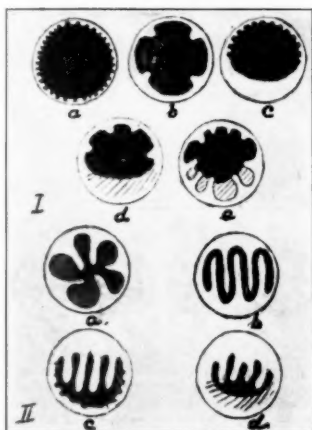


FIG. 2.—A series of transverse sections (I, a, b, c, d, e) of the lumen of the stomach, drawn from anatomical preparations, and showing how the lumen of the stomach with the same width of the muscular coat can vary considerably through different folding of the mucous membrane, with regard to the form as well as to the width of the inner surface of the stomach. Through such forms of contraction of the mucous membrane which are shown here—and many other combinations are further possible—the lumen of the stomach can either be formed into a channel with smooth sides or divided into different canals or systems of fissures between the lamellae. These can be placed in different ways (II, a, b, c, d, e), so that a filtering or sorting of the contents can take place, or small parts of the contents may be completely enclosed by mucous membrane in small digestion chambers.

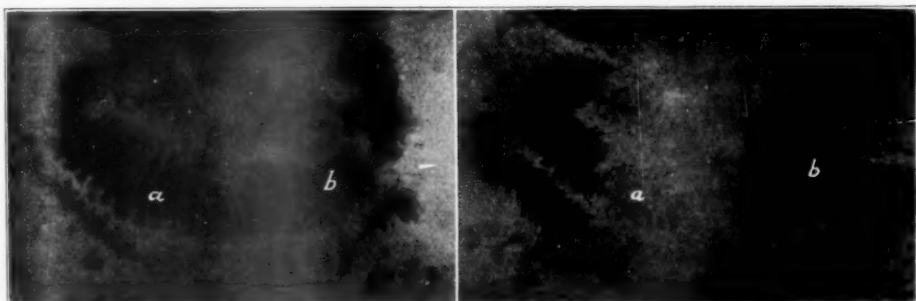


FIG. 3.—Two pictures of a high jejunal loop (ab), taken at an interval of one minute. Observe specially the area marked a. In the first picture there are in this area a few wide, simple and deep folds; in the second there are numerous close-set, small folds, dividing the lumen transversely into fine chambers.

the bulb, which were supposed to be due to local spasm of the muscular coat, were, in his opinion, mainly produced by deep and voluminous folding of the mucous coat.

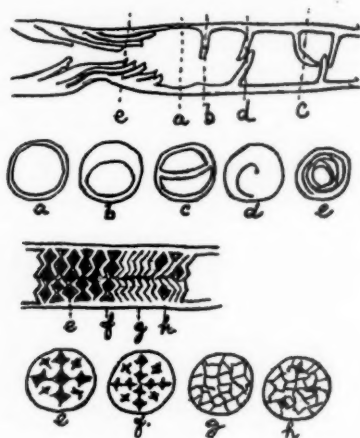


FIG. 4.—Showing, above, a longitudinal section of an intestinal loop where a number of different forms of simple mucous contraction are schematically outlined, and immediately beneath it, cross-sections of these varying folds: (a) indicates a fully free lumen, the loop forming an even tube; (b) a fold forming a valve to restrain the flow of the chyme; (c) and (d) folds forming bigger closed digestive chambers; (e) a separator-like formation, filtering the contents between the folds. The lower half of the figure shows a longitudinal section of an intestinal loop, where folds are refolded and in close contact with each other with corresponding cross-sections (taken at e, f, g, h) beneath. It is clear that by this complicated folding the intestinal contents are divided up into very fine digestive chambers surrounded by mucous membrane; the folds may fill the lumen entirely. All these forms of contraction may be found in radiograms of the living subject.

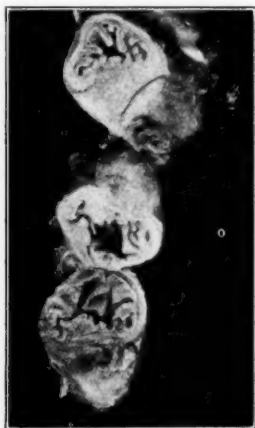


FIG. 5.



FIG. 6.

FIG. 5.—Showing three transverse sections of a strongly contracted sigmoid flexure. In the topmost section a muscular septum, originating from a transverse contraction of the muscular coat, fills the lower half of the lumen. On the other hand, the folds of the mucous membrane fill the remaining cavity in such a way that the section resembles a transverse section of the jejunum.

FIG. 6.—Radiogram of a strongly contracted sigmoid colon. The folds of the mucous membrane dominate the picture, as in the anatomical preparation shown in fig. 5.

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## Section of Epidemiology and State Medicine.

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### On some Outbreaks of Enteric Fever due to Carriers of Infection.

By FREDERICK DITTMAR, M.D.Glas., F.R.C.P.Edin.

(Medical Officer, Scottish Board of Health.)

(ABSTRACT.)

#### (I) ENTERIC FEVER IN A SCOTTISH POORHOUSE.

ON October 21, 1908, a man, aged 60, was admitted to the ordinary wards of the poorhouse (which is licensed for 168 beds, about forty of which are for lunatics). He was transferred to the sick ward on November 21, with symptoms of "endocarditis and pleurisy." At the latter date he also suffered from diarrhoea, which continued till after the beginning of January, 1909. The patient's bedding was often soiled with excreta, and washed in the institution laundry without previous disinfection. Suspicion of typhoid arose in February, and his blood was examined on February 16, 1909, when it gave a positive Widal reaction with three strains of *Bacillus typhosus*. But the organism of typhoid fever was *not* discovered in the urine or faeces. His case seems to have been an unrecognized one of enteric fever, and the other fifteen cases which occurred in January and February, 1909, among the inmates of the poorhouse, and of which only one arose among male lunatics, were at the time regarded as probably due to infection obtained originally through the laundry, where ordinary inmates do most of the washing. Three cases arose among the officials: the laundry woman, assistant matron, and sickroom night watchman. There were three laundry workers among the patients, one of whom was a male lunatic inmate, who died on February 14, 1909, before removal to the infectious diseases hospital. There were four deaths among the cases, or 25 per cent. mortality.

The outbreak was confined to the poorhouse, and milk and water could be excluded as sources of infection.

Of the cases, eight sickened between January 7 and 14, four between January 19 and 24, and three after January 24. One case had sickened earlier than all the others—viz., the man admitted on October 21, 1908, as an ordinary inmate, who had been removed to the sick ward on November 21.

No further information as to enteric fever in the institution was obtained

## 2 Dittmar: *Enteric Fever due to Carriers of Infection*

until April, 1914, when it was ascertained that five cases had occurred in the poorhouse since the last case in the outbreak early in 1909—viz., three cases among *male lunatic inmates* and two among ordinary inmates. The first was the case of a male lunatic inmate who had been in the institution for years, and who was discovered to be ill on April 17, 1909, and duly notified as enteric fever. He died on April 21, before removal to the infectious diseases hospital. It is to be noted that the lunatic inmate affected during the "outbreak" had died on February 14, or two months before this case was discovered.

After an interval of thirteen and a half months, another *male lunatic inmate* was discovered to have typhoid fever. He had been in the institution for years, was found to be ill on June 1, 1910, and removed to the infectious diseases hospital on June 6, where he died on June 9. The source of infection in his case was not ascertained at the time, but it was surmised that he might have been infected by drinking polluted water from a horse trough on the roadside near the poorhouse, and near which there was at times evidence of human excretal pollution. The man never worked in the laundry.

After an interval of two years and nine months a third case arose among the *male lunatic inmates*, a man, aged 53, who also had been in the house for years. His case was notified on March 22, 1913; he was removed to the infectious diseases hospital on the same day, and returned to the poorhouse after recovery.

The *two ordinary inmates* who had enteric fever in the poorhouse since the outbreak early in 1909 were both admitted with the disease, and transferred to the infectious diseases hospital.

The three male lunatic inmates who took enteric fever in April, 1909, in 1910, and in 1913 respectively, had been in the institution for years, and must have obtained their infection in the house from a carrier, or had it brought in to them. The latter method of infection could be excluded. Interest therefore centred on the possibility of finding the carrier.

On April 21, 1914, samples of blood were obtained from all (twenty-one) the male lunatic inmates and sent to the Royal College of Physicians Laboratory in Edinburgh for examination, and also to the Usher Institute of the University. As a result of the tests made, three cases, one of whom was known to have had typhoid fever, were regarded as possible carriers of the *Bacillus typhosus*, and samples of urine and faeces were obtained from each on April 27 for detailed bacteriological investigation. As a result, the organism was discovered in large numbers in the faeces of one of them—a dement, who had been for ten years in the institution. His blood gave a very high agglutinative reaction, namely, a complete reaction with 1 in 30, and 1 in 60, and a partial reaction in 1 in 120, 1 in 240, 1 in 480, and 1 in 960. A further specimen of this man's serum agglutinated the bacillus isolated from his faeces up to a dilution of 1 in 200. The patient was an old soldier, and may have had enteric fever while in the Army. He had been admitted to the lunatic department of the poorhouse on June 4, 1904, and his period of residence in the institution therefore covered all the outbreaks of enteric fever.

Subsequent to the discovery of the carrier, the following cases of enteric fever have arisen in the institution:—

On March 25, 1916, an ordinary inmate (who was semi-fatuous, though never in the lunatic wards) was found to be suffering from enteric fever, and removed to the infectious diseases hospital two days later. On investigation it transpired that he had been acting as pig-feeder, and had also helped the

inmate who carried the privy pails used by outside workers to the dungstead. This pail may at times have been made use of by the known lunatic carrier, and in this way infection may have been conveyed to the case in 1916. Three successive examinations of urine and faeces were made after this man's convalescence, and sent for bacteriological examination in July, August and September, 1916; all were reported as showing no evidence of any organisms of the typhoid group.

In March, 1918, after seven months' residence in the poorhouse, another male lunatic developed enteric fever, and was sent to the infectious diseases hospital. There can be no doubt that he was infected through the known carrier, who is a dement and very dirty in his habits. Every care is taken to keep him apart from others, and he has his own food dishes, &c., and sits at a special table to himself at meal times. But it is practically impossible to keep a case like this entirely separate from other people, even with the exercise of the greatest care.

In April and May, 1921, two cases of enteric fever arose in the poorhouse. The first was that of a lad, aged 17, who was an ordinary inmate, but, *inter alia*, cleaned brasses and w.c. seats in the male lunatic department. His infection was probably obtained in carrying out this work. He sickened about the last week of March.

The second case was that of the male lunatic attendant, who had held that post since December, 1913—a period of seven and a half years. He sickened in May, and was first seen by the doctor on May 16. His blood on that day gave a negative Widal reaction. On May 22 the reaction was positive, and he was transferred to the Infectious Diseases Hospital, where he died next day.

The points of interest in regard to enteric fever in this poorhouse are as under:—

(1) A male lunatic inmate, who was admitted on June 21, 1904, was discovered to be a carrier of the *Bacillus typhosus* in 1914—ten years later.

(2) From the time of his admission, and till January, 1909—a period of fully four and a half years—no known case of enteric fever arose in the institution.

(3) An outbreak which was confined to the poorhouse, and comprised fifteen sane cases and one insane, occurred early in 1909.

(4) A male lunatic inmate was found ill of enteric fever in April, 1909.

(5) A male lunatic inmate was found ill of enteric fever in June, 1910.

(6) A male lunatic inmate was found ill of enteric fever in March, 1913.

(7) Enteric bacilli were, in April, 1914, found in large numbers in the faeces of the male dement who was admitted to the male lunatic ward of the poorhouse on June 21, 1904.

(8) A male sane inmate who may have been infected through contact with enteric infection in a privy pail was found to have contracted enteric fever in March, 1916.

(9) A male lunatic was found to have contracted enteric fever in March, 1918.

(10) A male sane inmate took enteric fever in April, 1921.

(11) In May, 1921, the male lunatic attendant who had held that position for seven and a half years took enteric fever.

A male dement admitted in 1904 was proved to be a carrier of enteric infection in 1914. At times he was no doubt excreting enteric bacilli between June, 1904, when he was admitted to the lunatic department of the poorhouse,

#### 4 Dittmar: *Enteric Fever due to Carriers of Infection*

and January, 1909, when the first known case of enteric fever arose in the institution. There is no evidence that he contracted the disease after admission to the poorhouse, and it is highly probable that he had suffered from enteric fever during his period of military service in India. "Intermittency" is a very marked feature in this case. The record shows that a carrier of enteric infection (so long as he does not prepare food) may for long periods be in contact with his fellows without giving rise to cases.

#### (II) CIRCUMSCRIBED OUTBREAK IN A NORTHERN COUNTY.

In 1914 in a northern county in Scotland a circumscribed outbreak was traced to a farmer's housekeeper. Milk was produced from two cows at the farm and used in the household while the surplus was sold to a few people in the neighbourhood. The salient facts as to the cases were as follows:—

The cases arose in two households residing in separate houses about 800 yards apart in a direct line over intervening fields. Each house consisted of three apartments and in each family there resided the parents and three children. The only inter-communication between the families was that the children went to the same school; and both families obtained their milk from the same farm. There were no sanitary defects at the houses and there were no other cases at the school. Every other possible source of the disease, such as water, insanitary conditions at the houses, &c., could be excluded. The common milk supply was the only connexion between the cases which were notified as under:—

Date of notification	Initials	Age	Sex	Residence
(1) March 17, 1913 ...	A. B. ...	8 years 2 months	Female ...	Cottage A
(2) November 1, 1913 ...	A. F. ...	7 years 1 month	Male ...	" B
(3) May 3, 1914 ...	W. F. ...	5½ years	Male ...	" B
(4) June 20, 1914 ...	A. B. ...	11½ years	Male ...	" A
(5) June 24, 1914 ...	J. F. ...	40 years	Male ...	" B
(6) June 24, 1914 ...	E. F. (Mrs.) ...	35 years	Female ...	" B
(7) June 29, 1914 ...	M. D. (Mrs.) ...	36 years	Female ...	" A

The cows were milked by the housekeeper and the milk was entirely in her charge. The question of a carrier being the cause of the outbreak was gone into, and a specimen of faeces from the housekeeper at the farm was submitted to the bacteriologist at Aberdeen University. As a result of his investigation, the presence of *Bacillus typhosus* was reported in the sample submitted. A later investigation by another bacteriologist confirmed the first. There is nothing to show that this carrier ever suffered from typhoid fever, but as the disease is sometimes very mild, it may be assumed that she had passed through an attack of typhoid fever at one time. The fact that she harboured typhoid bacilli was proved, and since her departure from her employment no further cases have arisen in the district, which was naturally very free from enteric fever, only four cases having been recorded between 1902 and 1906, and none after that until the first of the series in this outbreak.

#### (III) OUTBREAKS TRACED TO A WORKING HOUSEKEEPER.

The next carrier of *Bacillus typhosus* was an unmarried woman, a working housekeeper. She had contracted enteric fever in the North of England, in October, 1913. The facts in regard to her case which raised the suspicion that she was a carrier, were as follows:—

In October, 1914, she went to act as temporary housekeeper to people living in an isolated cottage in a southern county in Scotland. The house-

hold consisted of a shepherd and his wife (who was ill, but *not* suffering from enteric fever) and an assistant shepherd. On November 17 the assistant shepherd sickened of enteric fever. A boy visitor from a neighbouring hamlet who had *one* meal in the house while the assistant shepherd was lying ill there took enteric fever, and his was the only case in the village from which he came. The cottage in which the family lived was new, with an excellent water supply and up-to-date drainage arrangements.

At the end of March, 1915, the suspected carrier went to act as housekeeper and cook in another family in a different part of the same county. The household consisted of two brothers, a hired lad, and an assistant herd. Early in May one of the two brothers sickened with enteric fever; and a fortnight later a man from a neighbouring cottage, who had some of his meals in the house during the lambing season, also sickened of enteric fever. At the end of May the hired lad left, and his place was taken by another who did not live in the house or have his meals there until the end of June, after it had been disinfected and cleaned and after the case of enteric fever who had been treated at home had recovered. This lad sickened of enteric fever in the latter part of September. During August and September the housekeeper milked two cows and supplied the milk and butter for the farm. The cook at the farm sickened of enteric fever.

Suspicion of the housekeeper being a carrier of enteric fever was aroused, and in June, 1915, samples of her blood and excreta were sent to the Laboratory of the Royal College of Physicians, in Edinburgh, for bacteriological investigation. On that occasion the result of examination of the excreta for *Bacillus typhosus* was negative. In October, 1915, arrangements were made to have fortnightly examinations made of the excreta in this case, and on October 28 the following report was obtained from the laboratory: "An organism with all the characters of the typhoid bacillus was obtained from the faeces of this case." The result of examination of a further sample of faeces sent on November 7, 1915, was negative.

The only conclusion that could be drawn was that the housekeeper was a carrier of enteric fever infection.

In this case further reports of the presence of typhoid bacilli in the faeces have been received from the Royal College of Physicians laboratory. These were reported as having been found in specimens of faeces on October 23 and December 8, 1915. For some weeks the local authority admitted this woman to one of their infectious diseases hospitals for treatment. After this and for over a year she acted as general servant at a children's home in the area of another local authority, and no cases of enteric fever arose among the children. But she was never allowed to handle milk or to bake scones; she acted as general servant and cut up vegetables, &c., for soup, but was not allowed to do anything with food further than prepare it for the pot. She carefully attended to all the precautions as to cleanliness of the hands and finger nails that were impressed upon her as being essential. She had her own towel and also her own nail-brush, and the matron of the home carefully supervised her in all her actions. The medical officer of health of the area in which she was acting as general servant in a children's home was officially advised of the presence of a known carrier in his district. Towards the end of 1919 two further specimens of excreta were sent to the Royal College of Physicians laboratory for bacteriological examination. The first specimens sent showed no typhoid bacilli. In September additional specimens were sent for examination, and typhoid bacilli were found in the faeces.

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This case presents what is not an uncommon feature in enteric carriers, viz., intermittency. The record also shows that (at least in an institution where scrupulous care is exercised) a carrier of enteric infection may be of no danger to others even when engaged to some extent in handling food.

### (IV) A URINARY CARRIER.

The next case was that of a private in the Army who was proved to be a urinary carrier. He was admitted to the City Hospital in Aberdeen suffering from typhoid fever in September, 1914, and it was proved that he had been infected by his wife in another town in Scotland. He was discharged from hospital at the end of September, 1914, after three successive examinations of his stools and urine had proved negative in regard to *Bacillus typhosus*. On discharge from hospital he went to his home in another town on a month's leave, after which he returned to his military duties in Aberdeen. Shortly after his return he began to complain of bladder irritation. This led to an examination of his urine being made and the discovery of *Bacillus typhosus* in the urine. He was readmitted to hospital for a course of treatment for his condition, and in April, 1915, he was discharged from the Army. Subsequently he returned to his home where he resumed his civilian occupation of a miner. Between April and June, 1915, three samples of his urine had been examined bacteriologically and each showed the presence of numerous typhoid bacilli. He was in good health and kept under regular observation by the medical officer of health who supplied him with urotropine as a urinary disinfectant. One of his children was removed to the infectious diseases hospital with typhoid fever in July, 1915. In January, 1916, bacilli were still demonstrable in the urine.

On December 27, 1916, the man's urine still gave fairly profuse cultures of *Bacillus typhosus*. About a year later the urine showed no bacilli of typhoid fever, and a further sample examined in February, 1921, was also reported as free from *Bacillus typhosus* by the bacteriologist.

This case may, I think, be regarded as "cured."

### (V) OUTBREAK IN A BURGH TRACED TO MILK INFECTED BY A CARRIER.

An outbreak of enteric fever in a small northern town was traced to milk produced by a carrier of the bacillus.

During October and November, 1920, some thirty cases of enteric fever in the burgh came to the knowledge of or were notified to the medical officer of health. Investigation showed that the majority of the cases had had the same milk supply. At the time of the outbreak most of the milk consumed in the town came from two sources, one local and the other imported, each of which supplied about an equal quantity to the inhabitants.

Of the cases fourteen had all and twelve part of their milk from the local dairy, while the other four cases had other sources of milk supply so far as could be ascertained.

The fact that two patients (who both died of the disease) obtained all their milk from the largest local farm before it was mixed with other milk at the dairy caused special attention to be directed to the workers there, when it was found that the blood of three female milkers gave a positive Widal reaction. The faeces and urine of one of them (whose blood gave an exceptionally strong Widal reaction) showed the presence of *Bacillus typhosus*. Everyone had been advised to boil all milk from the latter part of October, and all the milkers whose blood gave a positive Widal reaction were put off milking as soon as this was discovered. As a result of the measures taken the outbreak ceased.



## (VI) AN OUTBREAK IN A MENTAL HOSPITAL.

(a) *Time of the Outbreak.*

It was not till the end of May, 1921, that the true nature of some of the illnesses arising among inmates was recognized, as there had been no case of enteric fever in the institution for many years. On investigation of the outbreak, the question of illness of indefinite nature among inmates and staff not regarded at the time as of the nature of enteric fever was also gone into, and *inter alia* the post-mortem records were examined. As a result, three male and two female cases were added to those recognized during life.

From early in February till about the middle of September, a period of seven and a half months, fourteen cases of enteric fever sickened among the population of this asylum—thirteen among patients and one male attendant. Eight male and five female patients were attacked. Two males and a female patient (one male, enteric fever, and the female doubtfully so) sickened in February. Excluding the doubtful cases only one male patient sickened in February.

In March and April no cases fell ill.

In May, six cases, four male and two female patients fell ill.

In June, one female patient and one male (an attendant) fell ill.

In July, one male patient sickened; in August, one male; and in September, one female patient sickened. There have been no further cases.

The registered lunatics numbered 397 and there was a resident staff of seventy-five in the asylum, or 472 in all.

(b) *Inquiry into the Origin of the Outbreak.*

In May there were cases of undoubted enteric fever on both sides, male and female, of the asylum. The source of the disease was therefore common to both sides of the institution and possibly to the official resident staff as well, unless we assume that the head attendant was infected through the male cases of whom there had been several before he sickened in June. There is no coming and going between the two sides of the institution, inmates and attendants being rigidly confined to the male and female sides respectively, and also in the hospital. The two resident doctors of course see all inmates and sick members of the staff.

Common to everyone in the institution are: (1) The water supply, and (2) the milk supply. Common to all except to officials having their own houses in the grounds are: (3) Food prepared in the central kitchen; and (4) bedding, &c., washed in the steam laundry of the institution. In this connexion it may be stated that the bedding for female inmates is always kept entirely separate from that used for males.

Whatever its vehicle, the infection was not massive, but slight at any time, and sometimes appeared to be absent, e.g., in March and April; at one time rather more concentrated, e.g., in May, when six cases sickened; and at other times minimal in amount, e.g., in June, when two cases sickened, and in July, August and September, when one case fell ill in each month. Dealing now with each of the above possible common causes in turn I may say:—

*First, as to the Water Supply.*—This is from a source on high ground about one mile from the asylum, not liable to human pollution, and is slowly filtered through sand before distribution. The reservoir has a capacity of

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13 million gallons. Since the outbreak began, everyone (one attendant and ten male patients) in the water squad has been interrogated as to previous attacks of enteric fever or of illness suggestive of that disease, with negative result. In addition, specimens of blood from all have been tested for their agglutinative reaction to the typhoid group of organisms, with entirely negative result.

*Secondly, as to Milk.*—Milk is produced in the asylum dairy, and the workers, both official and inmate, have been the same for a long time. One milker (official) had been ill within the last few months with symptoms regarded as "influenzal" in nature. A specimen of blood obtained from her gave a negative Widal reaction. To set all doubt at rest, a specimen of blood was obtained from everyone (two milkers, one cattleman, and one patient) engaged in the dairy, and all gave a negative Widal reaction.

*Thirdly, as to the Kitchen Staff.*—All except one official had been working there for many months. A specimen of blood was obtained from everyone in the kitchen, and none gave a positive Widal reaction. In all, twenty-nine workers in the kitchen, both officials and patients, were examined.

*Fourthly, as to Laundry Workers.*—A specimen of blood was taken from every laundry worker (three official and fifteen patients) and examined for the Widal reaction. In every case the result was negative.

### (c) *Other Examinations made.*

Two inmates who suffered from diarrhoea had their blood examined for the Widal reaction on two occasions, at intervals of a week in each case, with negative result. In addition, specimens of faeces were examined from these cases for organisms of the typhoid group, with negative results in each. One of these patients died on June 25, and the post-mortem examination showed no evidence of enteric fever.

The Widal test was made in the case of all inmates whose temperature at the time when all patients' temperatures were being taken remained over 99° F. during twenty-four hours. There were five cases of this kind, and in four of them the reaction was negative; in the fifth (one of the recognized cases of enteric fever) the reaction was positive.

Everyone (inmates or staff) who had recently suffered from symptoms of an "influenzal" nature had specimens of blood examined for the Widal reaction. One of these (official) was engaged in the dairy, another was a male patient, and in both the Widal reaction was negative. A third was the asylum shoemaker, an ex-soldier, whose blood gave a positive reaction. He had never suffered from enteric fever, but he had had four antityphoid inoculations while in the Army. The first case among the inmates who died on February 20, 1921, had been working with him. The shoemaker's blood gave a positive Widal reaction on June 11. On June 17 a specimen of faeces from him was submitted to the bacteriologist, who reported on June 24 that no *Bacillus typhosus* was found in the specimen. Later, specimens of both urine and faeces were submitted for bacteriological investigation, and on October 14 both specimens were reported as showing none of the organisms of the typhoid group.

Of a total staff of forty-five attendants and nurses, three male attendants gave a history of having had typhoid fever at one time, viz.: One who had been in the asylum service since 1898 had typhoid fever many years ago. A second, who entered the asylum service on August 29, 1921, had suffered from the disease in 1919, and a third, who had been in the asylum since

July, 1919, had had enteric fever in Edinburgh in 1887. In the two last cases blood taken for the Widal test gave a negative result.

Three patients had been admitted from a parish in which there had been enteric fever early in 1921. One died on July 15, 1921, but during life there had been no clinical evidence of enteric fever, and after death there were no post-mortem appearances of the disease. From the second a specimen of blood was examined for the Widal reaction on October 13, with negative result. In the case of the third, who had been discharged from the Asylum, a specimen of blood from her also gave a negative Widal reaction to organisms of the typhoid group.

Shellfish and raw vegetables can be excluded as possible sources of the disease in any of the cases.

Of the tradesmen who bring food to the institution the baker's vanman was the only one who handled food that is not cooked before consumption. On inquiry he stated that he had had enteric fever nineteen years ago. But he did not begin delivering bread to the asylum till the end of June, several months after cases had arisen there. He could therefore be excluded as the source of the disease in the institution.

Lastly, the blood of all patients admitted since July 1, 1920, and still in the institution in December, 1921, and of all officials engaged since July 1, 1920, has been examined for the agglutinative reaction with negative result in every case. These comprised nineteen patients and eight officials.

This outbreak would appear to have been caused by some carrier of infection probably insane, whom it has not proved possible to trace by the investigations made so far.

#### (VII) THE ADMINISTRATIVE TREATMENT OF CARRIERS OF INFECTION.

The administrative treatment of carriers of infection has always been a difficult problem, and on January 21, 1921, the Scottish Board of Health issued regulations under the Public Health (Scotland) Act, 1897, for dealing with carriers of infectious diseases. The regulations came into force in Scotland on March 1, 1921. They give power to the local authority under the Public Health (Scotland) Act, 1897, to deal with a person adjudged to be a carrier of an infectious disease in the same manner as if he actually suffered from the disease. But before a person is to be deemed a carrier he must be certified as such by a medical officer of health and also by another registered medical practitioner, such certification to have effect for a period not exceeding three months. Further examinations may be made at any time after the date of a certificate, and the carrier may demand to be re-examined during the currency of a certificate on giving the medical officer of health not less than forty-eight hours' notice in writing.

Provision is also made for appeal to the Board by any person certified to be a carrier, and the Board may authorize one or more of their members to determine such appeal.

#### *Comment on the Regulations.*

Speaking generally, while the need for supervision in the public interest of the disease carrier is recognized, the liberty of the carrier has been guarded in the regulations.

This is no doubt necessary, but in the case of enteric carriers who form the most difficult class to deal with administratively, the time during which a certificate is valid seems to be too short. It is known that carriers of enteric

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infection may be so for years, and periods of thirty years and over are well authenticated in this condition. I would suggest that after the lapse of the first certificate in enteric carriers the period for re-certification should be yearly. But the most important disability of the carrier, most of whom are women, is their inability to earn a living as housekeepers, cooks, and dairy-workers. In the interest of others, carriers of enteric infection must not continue to do work that involves the preparation of food. If they *must* change their means of livelihood in the public interest, the charge of this should, I think, fall on the public purse. Each case would have to be judged on its merits and the individual circumstances and possibilities carefully investigated by the medical officer of health of the local authority concerned. If a public health local authority could certify, on the results of careful investigation into all the circumstances of the individual, that he or she could not earn a living at another form of occupation, then and only then would a disability pension be payable by the State to the individual so circumstanced, and it would be liable to revocation should the carrier on investigation prove to have lost his power of infectivity. It should also cease automatically on the carrier reaching the age of 70 years, when he would become entitled to an old age pension.

For the payment by the State of a disability pension to a carrier of infectious disease Parliamentary sanction would of course have to be obtained.

Dr. G. CLARK TROTTER said that Dr. Dittmar's paper was an excellent summary of results obtained by a large amount of tedious inquiry and research. One point in particular had struck him—namely, that of shortening, as far as possible, the tedious search for the carrier. In order to limit the number of Widal tests, the blood specimens had been taken from those considered most likely—for instance, in an asylum, the specimens had been derived from those most immediately in contact with the typhoid case occurring and the examinations had been very gradually extended until a positive result was obtained; so also with the confirmatory examination of faeces and urine for *Bacillus typhosus*. To examine *all* the asylum inmates and staff would be laborious and expensive; hence the short cuts. Now, taking an analogy from diphtheria, they all knew that swabbing of the general public would reveal many carriers; if examinations were thus also done extensively for the *Bacillus typhosus*, it might be found to be more prevalent than at present suspected, for the examinations for carriers, as he had said, had been limited to those most likely on the score of time and expense. When found, the search had been stopped. Might there not have been others unsuspected and undiscovered? They had to thank Dr. Dittmar for the examples drawn from these institutions, which were unique in some instances, owing to the apparent lull in the infectivity of the carrier which had retarded discovery.

## Section of Epidemiology and State Medicine.

President—Dr. R. J. REECE, C.B.

### Economics and Tuberculosis.

By R. J. EWART, M.D.

THE association of poverty and tuberculosis is well known, and much has been written on the subject. The difficulty has always arisen as to which of the two is the causative agent. To go into the literature is outside the scope of the present statement, but there is a general agreement that the two are highly associated. It is held by the Eugenic school, that inherent factors leading to poverty also carry with them a diathesis susceptible to lethal tuberculosis. It is not intended to controvert this statement, but simply to suggest that food, irrespective of diathesis, is a factor.

This relationship has been commented upon by Cobbett [1]. The Siege of Paris is cited as an example, when the rise of tuberculosis was very large, and the low rates for the following ten years are attributable to the sudden termination of the life of most of the tuberculous population at that time.

Austria and Russia of all continental countries showed the greatest reaction during the War, and there is no doubt that the economic stress was more keenly felt there than in any of the other belligerent countries. The conditions in Russia were of course too severe for comparison, as the present statement concerns itself with semi-starvation and not actual starvation. Further, in Russia the disorganization was such as to prevent any records being obtainable.

The following figures are quoted (i) from the *International Journal of Public Health*, September, 1920 [2]:—

#### DEATH-RATES, VIENNA.

Year	Death-rate	Phthisis
1912	15.5	—
1913	15.2	3.01
1914	14.6	2.86
1915	15.1	3.21
1916	15.2	3.56
1917	18.7	4.22
1918	21.0	4.26
1919	19.7	4.90
1920	25.3	5.58

In 1919: 63 per cent. of all deaths between 5 and 19 years in Vienna were due to tuberculosis as against 44 annually.

(ii) From the same Journal, February, 1920, No. 1, p. 93 [2a].

#### NUMBER OF DEATHS FROM TUBERCULOSIS IN VIENNA.

	1906	1913	1914	1915	1916
Civilian ...	7,427	6,376	6,158	7,014	7,810
Military ...	63	54	65	796	1,841
	7,480	6,430	6,223	7,810	9,651

Rösenfeld suggests that the distribution of the death-rates was modified by social circumstance. He showed that in the munition areas, the rates were not so high owing to high wages and better feeding.

The increase in institutional deaths in this country has also been attributed to the stringency of war-time conditions.

It has been suggested that the rationing schemes were more rigidly carried out, and the sense of hunger more scientifically appeased in institutions than among the population generally, and that non-nutritious fats and small quantities even of that, replaced the more wholesome articles. The point is suggestive and tends to support the view that food in its relation to real wages must have been a factor.

The statistical data I have used are mainly taken from papers by Mr. G. H. Wood [3]. Each year gives the mean real wages for the three previous years. The reason for this is, because the average period from notification to death is about three years, and it is assumed that the length of time between breakdown and death will be accordingly influenced.

The first column gives an index figure for the consumption of food and other necessities of life, and the second an index figure of the value of real wages.

The war period is given separately, as both consumption figures and real wages were subject to such special conditions that it is doubtful whether they are strictly comparable with those of the preceding years.

No figures are available before 1850 except Sauerbeck's wholesale prices, which begin at 1818, though for the earlier years no details seem to be given. These figures can be found in the *Journal of the Royal Statistical Society* as follows: 1818—45, September, 1886, p. 634; 1846—85, September, 1886, p. 648; 1882—1901, March, 1902, p. 87; 1895—1914, March, 1915, p. 281; 1902—16, March, 1917, p. 289.

These figures have been used by Dr. Stevenson [4] in the Registrar-General's report for 1919, and show a very close association between the sex ratio at birth and wholesale prices, and will be referred to later.

I think the general statement will be accepted that up to 1830 and onwards to 1850, the discovery of the practical uses of steam and its application to industry and production generally, was beginning to increase the work done by each individual, and hence the food available for consumption was beginning to increase. During this period phthisis exhibited its most pronounced and rapid decline. It may be accepted that if a population is 50 per cent. under-fed, the first 10 per cent. improvement will have a greater effect than the second 10 per cent., and so on. There may be a point, of course, when further increase in consumption might be followed by a fall in nutritional powers rather than an improvement; we have not reached this point as yet.

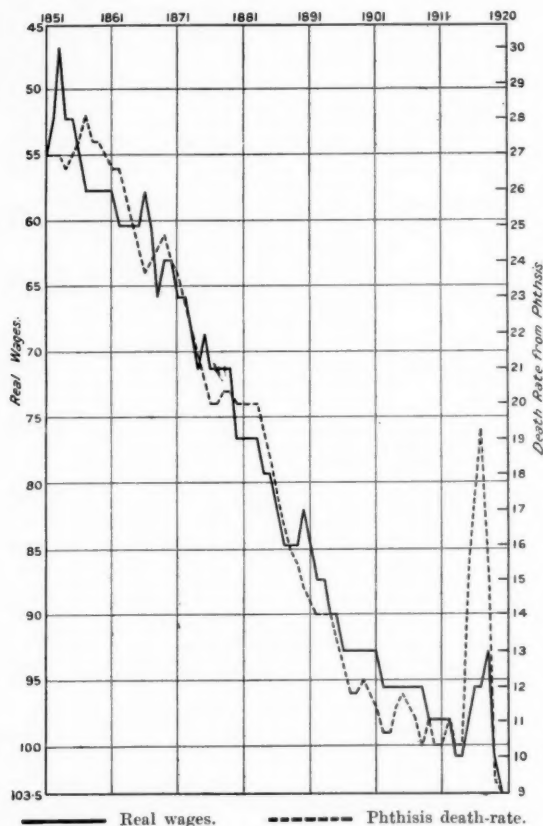
As to the condition of the working classes or rather the bulk of the population, the work of Charles Booth in East London, and of Rowntree in York, give a very reliable picture of the conditions existing at the close of the nineteenth century. For the years 1887-89, Booth gives 30 per cent. as living in poverty; Rowntree, using Atwater's standard and comparing family budgets, shows a rise and fall during an industrial life, and that over 30 per cent. (a similar figure) at some time or other received less food than the nature of the work demanded [5].

Turning now to the details of the conditions from 1914 to 1920, there is considerable difficulty in getting statistical figures which coincide with the experience and personal observations of those actually living through the period. The second Army Corps left England in November, 1914, and the authorities made very extensive requisition on the stocks of food available at the time. The more prosperous also began to buy extensively in view of a

Year	Consumption	Real wages	Death-rate from phthisis
1851 ...	—	55	27
1852 ...	—	55	28
1853 ...	—	55	30
1854 ...	—	56	28
1855 ...	—	55	28
1856 ...	—	54	27
1857 ...	—	52	26
1858 ...	—	54	26
1859 ...	—	54	26
1860 ...	84	55	26
1861 ...	84	56	26
1862 ...	81	56	25
1863 ...	78	58	25
1864 ...	83	60	25
1865 ...	85	62	25
1866 ...	87	64	26
1867 ...	89	63	25
1868 ...	88	62	23
1869 ...	89	61	24
1870 ...	92	63	24
1871 ...	94	64	23
1872 ...	96	66	23
1873 ...	98	68	22
1874 ...	100	70	21
1875 ...	102	72	22
1876 ...	102	74	21
1877 ...	102	74	21
1878 ...	103	73	21
1879 ...	103	73	21
1880 ...	103	74	19
1881 ...	102	74	19
1882 ...	102	74	19
1883 ...	104	74	19
1884 ...	105	76	18
1885 ...	105	78	18
1886 ...	104	81	17
1887 ...	104	83	16
1888 ...	104	85	16
1889 ...	106	86	16
1890 ...	109	88	17
1891 ...	113	89	16
1892 ...	115	90	15
1893 ...	113	90	15
1894 ...	112	90	14
1895 ...	112	92	14
1896 ...	115	94	13
1897 ...	117	96	13
1898 ...	118	96	13
1899 ...	118	95	13
1900 ...	120	96	13
1901 ...	119	97	13
1902 ...	119	99	12
1903 ...	118	99	12
1904 ...	117	97	12
1905 ...	117	96	12
1906 ...	118	97	12
1907 ...	120	98	12
1908 ...	119	100	12
1909 ...	119	98	11
1910 ...	119	100	11
1911 ...	120	100	11
1912 ...	122	98	11
1913 ...	124	100	10
1914 ...	—	—	10
1915 ...	—	—	11
1916 ...	—	—	12
1917 ...	—	—	12
1918 ...	—	—	13
1919 ...	—	—	10
1920 ...	—	—	9



possible shortage. A popular feeling at the same time tended to curtail the rise of prices. It so happened, that during the Christmas season, 1914-15, although wages and prices had not changed materially, there was a shortage in the amount of food available for the working-class population. This is shown remarkably well in the series of figures given in the number of children fed at the school feeding centres.



Estimate of movements of time and piece rates in certain industries compared with the change in the cost of living, gives the following:—

July		Wages general Rough average		Cost of living Labour Gazette index		Modified index
1914	...	100	...	100	...	100
1915	...	105-110	...	125	...	120
1916	...	115-120	...	145	...	135
1917	...	135-140	...	180	...	160
1918	...	175-180	...	205	...	180
1919	...	210-215	...	210	...	185
1920	...	260	...	252	...	220

This gives an index of real wages:—

					Phthisis
1914	...	...	100	...	1.02
1915	...	...	86	...	1.43
1916	...	...	81	...	1.16
1917	...	...	76	...	1.23
1918	...	...	86	...	1.32
1919	...	...	103.5	...	0.98
1920	...	...	103	...	0.87

FREE MEALS PROVIDED TO SCHOOL CHILDREN.

		Dinners		Children fed
1921-22	...	60,676,017	...	592,518
1920-21	...	10,447,596	...	148,082
1919-20	...	6,335,821	...	75,013
1918-19	...	5,647,954	...	53,742
1917-18	...	6,518,174	...	60,633
1916-17	...	5,777,147	...	65,301
1915-16	...	9,957,634	...	118,114
1914-15	...	29,560,316	...	422,401
1913-14	...	14,525,593	...	156,331
1912-13	...	19,001,729	...	358,306

The first table is taken from Professor Bowley's work [6], according to which the position of real wages fell at first and then improved until the position in 1920 was at about the level of the pre-war period or slightly above it. The number of children fed at school agrees with these figures and reflects rather more accurately what was actually happening.

The phthisis death-rate followed the same trend up to 1916; 1917 and 1918 were of course disturbed by the epidemics of influenza, which as we know increased the rates, and also by anticipating the prospective deaths, reduced the rates for 1919. Many consumptives who might have lived for twelve or eighteen months were probably killed then. This fact was also statistically verified in one area, by tracing the deaths to families of known diathesis.

Thus the war period as far as judged by the supply of free meals, shows not only the relationship of phthisis death-rate to the supply of food but also indicates that the response is rather more rapid than as measured by a mean of the previous three years and may be measured from year to year.

In 1914 the percentage based on Mr. Booth's standard of poverty may be estimated to have fallen to between 10 and 15 per cent. (mal-nourished school children). It cannot of course be stated that the whole population is even now adequately fed; but the movement of index figures, giving average consumption or value of real wages, is highly correlated with changes in the proportion of half-starved people living at any particular period.

That a more suitable diet should enable some to acquire an immunity to tuberculosis and prolong the period between the infection and death is easily understandable. Some doubt may be expressed also whether this is the sole factor, and considerations relative to "inherited diathesis" lend credence to the belief in biological factors outside human agency. Amongst such biological agencies is the possible influence of the age of the parent at the time of the birth of the child upon the physical and mental qualities of the child. In a series of contributions to the *Journal of Hygiene* [7] I have collected evidence tending to show the reality of such influence and I suspect that it is also exerted in the particular case of power of resistance to tuberculosis.

On the hypothesis (a) that liability to breakdown from tuberculosis is a function of malnutrition, (b) that the offspring of elderly parturients are worse nourished in embryonic life than those of young parents and that this effect persists in post-natal life, we should find that the age at which a subject breaks down is negatively correlated with the parent's age at the birth of the

subject, due allowance being made for other factors. It is impossible to test this adequately. The attached table shows that the total correlation is almost zero but other factors are *not* constant. If we could regard the age at death of the mother as a measure of the remaining variable factors and make suitable allowance, a negative correlation would be found which may have some significance. (This has been roughly done and the result seems to support the suggestion made.)

The suggestion therefore is that some such effect as postulated may exist, but the data are far too scanty and too little homogeneous for much weight to be attached to this particular result.

DISPENSARY POPULATION OF PULMONARY TUBERCULOSIS 15-46. EAST HAM, ILFORD, AND BARKING.

(All advanced cases or those in which tubercle-bacilli found in sputum.)

Age of Mother at Birth.

Age of attack	15-16	17-18	19-20	21-22	23-24	25-26	27-28	29-30	31-32	33-34	35-36	37-38	39-40	41-42	43-44	45-46	Totals
13-16 ...	—	—	—	1	2	1	3	4	2	—	2	—	—	—	—	—	15
17-20 ...	—	1	1	1	2	5	3	5	10	3	3	3	2	1	—	—	40
21-24 ...	—	—	—	1	6	1	5	2	4	2	1	1	3	1	1	1	29
25-28 ...	—	—	4	2	6	5	3	2	3	3	3	—	1	1	1	—	34
29-32 ...	—	—	2	2	3	7	2	6	4	6	2	3	1	—	—	—	38
33-36 ...	—	—	1	1	7	8	5	6	6	3	6	4	3	1	2	1	54
37-40 ...	1	—	1	2	2	1	4	1	5	2	4	1	2	1	2	—	29
41-44 ...	—	—	—	2	1	4	1	2	1	1	1	1	1	—	—	—	15
45-48 ...	—	—	1	2	—	—	1	2	2	1	—	2	2	—	—	—	13
Totals ...	1	1	10	14	29	32	27	30	37	21	22	15	15	5	6	2	267

Standard deviation—age of attack, 6.48 years.

Born before 30 years—6.46.

" " " " mother at birth, 7.18 years.

" after 30 " —6.50.

Co-efficient of correlation = + 0.031 ± 0.05.

Note.—At time of writing the majority are dead and died of tuberculosis.

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- [6] BOWLEY, Professor, "Wages and Cost of Living" (1914-20), p. 106.
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#### DISCUSSION.

Dr. MAJOR GREENWOOD said that Dr. Ewart's paper fell into two parts, the first dealt with facts, many of which were notorious; but Dr. Ewart had handled these facts in an interesting way. He (Dr. Greenwood) supposed that the ætiological importance of under-nutrition was admitted by all who had studied the subject, but he assumed that there would not be unanimity with respect to some of Dr. Ewart's inferences from his charts. In particular the inference that the assistance rendered by Boards of Guardians in times of bad trade effectively compensated, so far as national nutrition was concerned, the loss of family income seemed to him far too good to be true. He should have thought it much simpler to suppose that the lack of correspondence between the employment and death-rate graphs was a resultant of many other factors—including a very low order of accuracy of the former. From the statistical point of view, he should hesitate long before drawing conclusions of a precise nature from time charts. The fact that the rate of mortality from tuberculosis was falling faster before the discovery of the bacillus of tuberculosis than since, while a conclusive proof that the

discovery of the pathogenic *materies morbi* was not an essential preliminary to sanitary action—not indeed that any such proof was needed—was no proof at all that measures based upon pathological work were unsound. It was as unreasonable to sneer at bacilli as to worship them. The second part of Dr. Ewart's paper propounded a very interesting hypothesis, viz., that resistance to tuberculosis was partly dependent upon the age of the parents at the time of reproduction. The actual statistical evidence in favour of this hypothesis contained in Dr. Ewart's paper was admittedly scanty—one might say *very* scanty. At present all one could do was to wish Dr. Ewart success in investigating a very difficult but very interesting problem.

Dr. S. VERE PEARSON first referred to the writings of Dr. Bertram G. M. Baskett, which appeared chiefly in the correspondence columns of the *Lancet* and *British Medical Journal* a few years ago. These entirely corroborated the view regarding real wages and the incidence of tuberculosis which Dr. Ewart put forward, and they included a survey of statistics dating further back than Dr. Ewart's, and embracing other countries than our own. One of Dr. Baskett's articles, and an important one, appeared in the *Yorkshire Post*, June 4, 1919. By quoting two sentences from this he (Dr. Pearson) wished to bring out certain aspects of the subject before the meeting which he thought did not receive the attention their importance demanded. Dr. Baskett wrote: "All the time real wages were rising, taxation, direct and indirect, was falling, often absolutely, always relatively to the growth of wealth." And " . . . Government and municipal measures are increasing poverty and emphasizing the maldistribution of wealth." In the article from which this quotation was made Dr. Baskett showed that the employers' and even probably the State's contributions under the Insurance Act were passed on to the employees. Similarly most would probably agree that indirect taxes, such, for example, as the heavy breakfast table duties, were real burdens upon the poor. But many so-called direct taxes, Dr. Pearson believed, were also passed on, often in subtle ways, to the workers. Now that taxes in this country were at least five times what they were in 1913, it was expedient to study their true incidence, particularly in relationship to the value of real wages and the death-rate from tuberculosis. If present methods of taxation increased poverty, then, even though some of the funds collected were spent on schemes for combating tuberculosis, probably they made preventable disease more rife.

The tendency of the day in most lands was to an increasing army of bureaucrats, and in some countries it was becoming a question as to whether the general body of producers could carry the large body of civil servants of one sort or another which had risen to serve them. This led him to another point: most of the efforts put forward by those engaged in working the various antituberculosis schemes were directed to tending the sick, a very necessary and humanitarian proceeding, but not a truly preventive one. Even the tuberculosis officer, originally supposed to examine contacts, to visit and help to improve the environment, and to educate the people into paths of better hygiene, still had far too much of his time taken up by treating the sick. The adage that prevention and cure went hand in hand together was incorrect and harmful. Preventive measures were largely political matters, they should be recognized as of interest to doctors as citizens rather than as medical men and women.

Dr. CHALMERS thought that Dr. Ewart had done well in submitting his subject in a form which would readily create a wide field for discussion. It was, of course, an old view that the progress of the working classes was to be gauged rather by the increase in the purchasing power of wages than by the actual increase in the amount. Sir Robert Giffen, he thought, had made that clear. His own view was that a considerable part of the reduction in the tuberculosis and general death-rates which set in so definitely in Scotland at least, in the early seventies, was related to the commencing and ultimately widespread importation of foreign meat, and it would be a fair speculation to continue the argument further so as to include as a late effect of this increased food supply the reduction in the infantile death-rate which only began in a definite way during the present century in a generation which was itself the child of that which, in its adolescence, had benefited by the first rush of imported food. This might

be regarded as illustrating the effect of an increased food supply on the somatic cell; and the paper had a definite value at a time when so large a proportion of the people were unemployed, and there was risk of extensive underfeeding.

Dr. F. J. H. COUTTS, in view of Dr. Ewart's emphasis on the relation between the rise in real wages and the decline in tuberculosis, expressed surprise at the opinion set forth by Dr. Ewart that periods of serious unemployment did not affect the death-rate from tuberculosis. It also appeared to him (Dr. Coutts) doubtful whether the assistance given by Boards of Guardians in such periods of unemployment could sufficiently account for this. Dr. Coutts agreed with the view which had evidently received general assent that tuberculosis was very largely a problem of nutrition, and referred to the experience during the War in Poland and Austria, where the death-rate from tuberculosis had enormously increased, when, owing to war conditions, the nutrition of the population was seriously affected. Boards of Guardians could scarcely give assistance to such an extent as to bring the income of the unemployed up to the standard of those employed. He noted the large increase in unemployment in the latter part of 1921, and feared that this would probably be followed by an increased mortality from tuberculosis in the present year. The figures for the first six months of 1922 were not yet available, but the mortality statistics for the first quarter of 1922 indicated that there was a considerable increase in tuberculosis mortality over that of the first quarter of 1921.

Notwithstanding the undoubted connexion of the decline of tuberculosis with the rise in real wages and the related improvement in general nutrition associated with this rise, he was of opinion that other factors were also concerned with the reduction in tuberculosis. He regarded the general sanitary improvement during the nineteenth century an important contributory factor along with the better education of the public in matters of hygiene. He thought that if, instead of considering figures for the whole country, the statistics of separate areas could be considered, it would be found that certain areas, in which energetic and precise special measures had been taken with regard to tuberculosis, had shown a greater decline in the incidence of tuberculosis than others in which similar intensive efforts had not been made. In connexion with a disease like tuberculosis, many factors had to be brought into consideration, and it would not be wise to attribute changes to one factor alone.

Dr. HAMER said there seemed to be a tendency, nowadays, to deprecate insistence upon the fact that the rate of decline of phthisis began to slacken soon after Koch discovered the bacillus. They were admonished not to speak disrespectfully of bacilli. While, however, no one surely could underrate the positive value of the work of the last twenty or thirty years, it was impossible to ignore the phenomenon in question. Bulstrode, when he drew up his chart of death-rates in towns, filled them up to the year 1905, leaving blank years up to 1910, in order, as he used to say, that the filling in of the blank spaces might drive home the fact that the rate of fall was declining. A few years later Professor Karl Pearson showed that Bulstrode's forecast was correct. It all seemed so simple now, but it was just those very simple things that required an eye like Bulstrode's to see them. It was difficult for most of us clearly to appreciate in advance that, if a man died in a sanatorium in this country, he could not also die in America, or South Africa, or Australasia.

Dr. R. J. EWART (in reply) said that the experience of Paris after the siege suggested that any rise in the death-rate from tuberculosis during the current year might be dependent on the same cause and not be due to the prevalent wave of industrial depression. The curves of unemployment for previous years did not suggest any such association. The data dealing with the influence of parental age were purely tentative, the difficulties were mainly two: first, the doubt as to the nature of a dispensary population and secondly the statistical difficulty of assessing the influence of age of parent at death. Many of the parents were at the time of observation still alive, and hence an age had to be taken such as would divide them into two classes: (a) those who were all dead; (b) some still living and some dead. One of the squares of the fourfold table so formed must necessarily be blank. The value of the correlation co-efficient under such conditions must necessarily be of doubtful significance.

## Section of Epidemiology and State Medicine.

President—Dr. R. J. REECE, C.B.

### On the Age and Sex Distribution in Scarlet Fever.

By F. M. TURNER, M.D.

THE annual reports of the Metropolitan Asylums Board contain a large number of tables showing the total number of scarlet fever admissions, divided according to age and sex. Such tables were published regularly each year from 1887 till 1914. The table for 1914 is given below, as a sample.

TABLE I.—METROPOLITAN ASYLUMS BOARD. SCARLET FEVER ADMISSIONS AND DEATHS DURING 1914, DIVIDED ACCORDING TO AGE AND SEX.

Ages	Males		Females		Total	
	Admitted	Died	Admitted	Died	Admitted	Died
Under 1 ...	97	6	76	5	173	11
1-2 ...	348	18	351	15	699	33
2-3 ...	618	20	602	21	1,220	41
3-4 ...	808	31	817	26	1,625	57
4-5 ...	1,019	22	1,107	16	2,126	38
5-10 ...	4,317	27	4,966	43	9,283	70
10-15 ...	1,877	15	2,406	7	4,283	22
15-20 ...	568	8	646	5	1,214	13
20-25 ...	242	4	341	—	583	4
25-30 ...	148	4	247	3	395	7
30-35 ...	79	2	150	3	229	5
35-40 ...	44	1	61	—	105	1
40-45 ...	14	—	31	1	45	1
45-50 ...	4	1	10	—	14	1
50-55 ...	3	—	2	—	5	—
55-60 ...	1	—	3	—	4	—
Over 60 ...	1	—	2	—	3	—
Total ...	10,188	159	11,818	145	22,006	304

All these tables are of the same form except one, that for 1887, in which the first age-group is from 0-4 years inclusive.

Similar age and sex tables for the cases notified were published in the report for 1895 of each subsequent year till 1914.

Details for the war years were not published but the table was resumed in 1920. By the courtesy of the chief office I have been furnished with the figures for the five missing years.

In the first series of tables there are over 350,000 cases tabulated, and in the other over 430,000. Of course a large number of cases are common to the two series.

On looking at so large a mass of material the first idea to strike one is that each year is a repetition of its predecessors, not with mathematical accuracy, but with such small variations as chance always produces. A meeting was held a year ago between a few of the Metropolitan Asylums Board medical officers and other statistical experts, at which I was present, in order to discuss improvements in the form of various tables in the annual reports. At that

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meeting it was decided to resume the publication of the age and sex tables with slight alterations. If the variations from one year to another are so slight as to be within the limits of chance variations, it is obvious that nothing further can be gained by continued publication of such statistics. On the other hand, if the variations from one year to another exceed the probable limits of chance variations, they represent a natural phenomenon in some way connected with the disease. The significance may not be plain, but the continuation of the tables in future years may throw light on what is now obscure.

On each point in which I have examined these tables I find the variations far greater than chance can account for, and this paper records the results.

### (I) MEAN AGE IN MALES AND FEMALES.

Table II gives the number of males and females admitted each year; Table III the mean age for each sex in each year. The tables also show the number of cases of each sex notified in each year and the mean ages of these cases.

TABLE II.—SCARLET FEVER. NUMBER OF CASES IN LONDON.

Year	Cases notified			Cases admitted to Metropolitan Asylums Board hospitals		
	Males	Females	Ratio 1 to	Males	Females	Ratio 1 to
1887	...	...	...	2,902	2,998	1.033
8	...	...	...	2,129	2,279	1.070
9	...	...	...	2,111	2,407	1.140
1890	...	...	...	3,115	3,422	1.098
1	...	...	...	2,447	2,815	1.150
2	...	...	...	6,188	6,905	1.115
3	...	...	...	7,047	7,501	1.064
4	...	...	...	5,543	6,055	1.092
5	9,421	10,177	1.080	5,495	5,776	1.051
6	12,226	13,275	1.085	7,715	8,267	1.073
7	10,687	12,015	1.124	7,265	7,848	1.080
8	7,984	8,816	1.104	5,880	6,245	1.062
9	8,515	9,484	1.114	6,397	6,893	1.077
1900	6,530	7,260	1.112	5,138	5,245	1.013
1	8,837	9,543	1.080	7,084	7,455	1.052
2	8,844	9,408	1.064	7,135	7,368	1.032
3	6,060	6,468	1.062	5,046	5,299	1.050
4	6,375	7,064	1.108	5,362	5,793	1.080
5	9,142	10,319	1.118	8,021	8,937	1.114
6	9,643	10,686	1.108	8,475	9,458	1.116
7	12,101	13,824	1.142	10,699	12,065	1.128
8	10,377	11,694	1.136	9,371	10,258	1.095
9	8,245	9,009	1.093	7,396	7,988	1.080
1910	4,990	5,519	1.106	4,191	4,591	1.096
1	4,833	5,650	1.169	4,093	4,725	1.154
2	5,287	6,034	1.141	4,654	5,229	1.123
3	7,952	9,591	1.206	6,755	8,255	1.222
4	11,580	13,468	1.163	10,188	11,818	1.160
5	7,958	9,145	1.149	...	...	...
6	3,998	4,760	1.190	...	...	...
7	2,766	3,371	1.218	...	...	...
8	2,997	3,853	1.285	...	...	...
9	5,810	7,143	1.230	...	...	...
1920	9,989	12,730	1.274	...	...	...
Total	203,147	230,306	1.133	167,842	183,855	1.095

It will be seen that the mean age of females notified is always higher than that of males notified in the same year; the difference remains fairly constant from year to year in spite of fluctuations in each column taken separately: it shows however a definite increase in the later years. Between 1895, and



1905 it is usually about 0·5 of a year; from 1906-14, about 0·7; and from 1915-20, from 1·0 to 1·3.

If we examine the mean age of cases admitted we find a general resemblance, with certain differences. In one year, 1900, the mean age of males exceeded that of females by 0·302; in all other years the mean age of females is the greater. The difference is, however, not quite so great as with the notified cases. From 1887-1903 it is usually about 0·3, but there are two or three exceptions; from 1904-14 usually from 0·4 to 0·6. Comparing the two sexes, therefore, we can consider the greater mean age of females an almost constant phenomenon, but somewhat greater in the later years. Perhaps the figures for 1915-20 have in some way been influenced by exceptional war conditions.

TABLE III.—MEAN AGE OF SCARLET FEVER PATIENTS, LONDON.

Year	Cases notified			Cases admitted to Metropolitan Asylums Board hospitals		
	Males	Females	Difference	Males	Females	Difference
1887	...	...	...	8·775	9·123	+·348
88	...	...	...	8·681	9·055	·374
89	...	...	...	7·840	8·634	·794
1890	...	...	...	8·378	8·568	·190
91	...	...	...	8·340	8·882	·542
92	...	...	...	8·863	9·242	·379
93	...	...	...	9·058	9·327	·269
94	...	...	...	8·437	8·745	·308
95	8·624	9·125	+·501	8·366	8·754	·388
96	8·673	9·258	·585	8·560	8·897	·337
97	8·444	8·905	·461	8·327	8·546	·219
98	8·533	8·951	·418	8·509	8·738	·229
99	8·780	9·356	·576	8·630	9·069	+·439
1900	9·528	9·760	·232	9·287	8·985	—·302
01	9·141	9·667	·526	9·030	9·166	+·136
02	8·833	9·380	·547	8·559	8·870	·311
03	8·499	9·075	·576	8·186	8·526	·340
04	8·124	8·916	·792	7·808	8·461	·653
05	8·119	8·713	·594	7·799	8·229	·430
06	8·452	9·203	·751	8·336	8·744	·408
07	8·610	9·257	·647	8·577	8·965	·388
08	8·616	9·483	·867	8·596	9·189	·593
09	8·762	9·514	·752	8·569	9·117	·548
1910	8·521	9·323	·802	8·310	8·870	·560
11	8·574	9·309	·735	8·390	8·976	·586
12	8·546	9·205	·659	8·294	8·764	·470
13	8·984	9·729	·745	8·780	9·300	·520
14	9·171	9·996	·825	8·844	9·508	+·664
15	8·866	9·893	1·027	...	...	...
16	8·619	9·779	1·160	...	...	...
17	8·695	9·729	1·034	...	...	...
18	8·364	9·670	1·306	...	...	...
19	8·720	9·947	1·227	...	...	...
1920	9·064	10·296	+1·232	...	...	...

Taking each sex separately we have, for males notified, the lowest mean age, 8·119 in 1905, and the highest 9·528 in 1900. For males admitted the lowest, 7·799 in 1905, and the highest, 9·287 in 1900. For females notified, 8·713 in 1905, to 10·296 in 1920. For females admitted, 8·229 in 1905, to 9·508 in 1914. The range of variation is, therefore, considerably over one year in each column, and in one column is over 1·5.

Before considering the significance of these variations, we must examine the effects of chance. Theory shows that of chance variations in any series about one-half will fall within the limits  $M. \pm P.E.$ , where  $M.$  is the mean value and  $P.E.$  its probable error or probable deviation. Also that less than one-twentieth will fall outside the limits  $M. \pm 3 \times P.E.$

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To calculate the P.E. for each of the 108 figures of Table II would require a greater labour than the calculation of the means; a simpler method will suffice. The P.E. varies inversely as the square root of  $N$ , the number of cases included in each calculation of the mean. It is, therefore, high in 1917—males P.E.  $\pm 0.082$ , females, 0.083; and low in 1914 and 1920, males 0.41 both years; females 1914, 0.041; 1920, 0.042. We make no error, therefore, in assuming that all the probable errors of columns one and two are less than the values of the year 1917. Taking the twenty-six figures in column one, the mean of which is approximately 8.70, six of them differ by less than 0.082, five more by less than double this figure and so on. The complete table of deviations in terms of probable errors for the two sexes is as follows:—

Deviations	Number of cases	
	Males	Females
< P.E. ....	6	6
> P.E. < 2 P.E. ....	5	3
> 2 P.E. < 3 P.E. ....	5	3
> 3 " < 4 " ....	3	0
> 4 " < 5 " ....	2	7
> 5 " < 6 " ....	2	2
> 6 " < 7 " ....	0	1
> 7 " < 8 " ....	2	2
> 8 " < 9 " ....	0	1
> 9 " < 10 " ....	0	0
> 10 " < 11 " ....	1	0
> 11 " < 12 " ....	0	1
Total ...	26	26

It is evident, therefore, that chance variations cannot account for the observed fluctuations which are approximately three times too large for such an origin. Allowing for the much smaller probable error in epidemic years, the fluctuations are about five times larger than if chance alone had been their origin.

Another argument leads to the same conclusion. Fluctuations due to chance would be irregularly distributed; any regularity observed tends to prove a different origin. Now the fluctuations of the mean tend to show two forms of regularity. First, high and low values tend to occur in groups two or three together, and secondly the values for the two sexes in most cases rise and fall together. Calculating the correlation between the values for males and females in the same year  $r = 0.71$ , a fairly high correlation. If chance had caused the differences it is difficult to avoid the conclusion that the figures for the two sexes in any one year should be quite independent. Admitting, then, that the fluctuations are for the most part too large to have a chance origin, can we find any evidence to point to any general law? In column two we find all the figures after 1913  $> 9.7$ , and with one exception all figures before 1913 less than 9.7. There is therefore a slight tendency to increase of mean age as we pass from the earlier to later years. If however we leave out 1915 and subsequent years as possibly abnormal, this tendency becomes much less pronounced.

Apart from a steady increase through the whole table we find well marked oscillation from low figures to high and back again. Thus 1887, 1892, 1893, 1900, 1901, 1913, 1914 and 1920 show high mean ages in both sexes; 1889, 1890, 1904, 1905, low means.

Now Table II shows that the incidence of scarlet fever in London fluctuates

greatly from year to year, as a rule three or four high years are followed by three or four low, and since 1903 a very regular seven-yearly oscillation is shown. 1907, 1914, and 1921 (not shown in the table) form crests of the waves, and 1903, 1910, 1917 troughs. The preceding figures are not quite as regular. 1887, 1893 and 1896 were epidemic years, in 1889, 1895 and 1900 the prevalence was low. Carrying backwards the seven-year period would give crests at 1886, 1893 and 1900, corresponding well in two instances but not at all in the third. It therefore seems more than a coincidence that the years of high prevalence, 1887, 1892, 1893, 1914 and 1920, should show high mean ages; and 1889 and 1904, years of low prevalence, low means. A striking exception is 1900 when a low prevalence coincided with a high mean age in both sexes. It may be significant of some underlying cause that 1900 should have been an epidemic year if the seven-year wave had been regular.

There are several instances in which the causes of prevalence and of mean age do not quite coincide but reach a crest or a trough at an interval of one or two years. Thus, very low mean ages occur in 1904 and 1905, low prevalence in 1903. Low mean age in 1918, low prevalence in 1917 and 1918.

Taking all the figures for cases notified, I find a correlation  $r = 0.17 \pm 0.13$  for number of male cases notified and the mean age of males; and a correlation of  $r = 0.13 \pm 0.13$  for females. This looks as if the connexion between these two factors was slight or none. I am however inclined to believe that it is real in view of the facts given above, and that statistics of future years will show the connexion.

If we compare the mean ages of cases notified and cases admitted during any one year, the former is always the greater. The difference in males ranges from 0.020 to 0.327, in females from 0.213 to 0.765. The fact that the difference is always in one direction is sufficient proof that it is not accidental. It is also much larger than the P.E. for any of the figures.

It is difficult to find the true explanation of this. If the only difference between the two sets of figures was that certain cases are nursed at home, the difference would show a selection of cases according to age, on the whole a larger proportion of old than of young patients being retained. However the facts are a little more complex. The proportion of London cases admitted to cases notified in 1890, was 43 per cent. It rose steadily to 80 per cent. in 1902, and 90 per cent. in 1908. Since then it has more often been above than below 90 per cent. There are however two other causes for divergence between the two series of figures with which we have been dealing: (1) A small number of extra-metropolitan cases are admitted every year, chiefly from Tottenham. In 1914 these numbered 400, in 1920, 348. (2) Cases admitted are subject to a revision of the diagnosis, which of course does not take place in the notifications. Thus in 1914, 1,227 cases were admitted with certificates that they were suffering from scarlet fever, and were not found to be so suffering, in 1920 there were 931.

It is tempting to believe that the discrepancy is due to a difference in age-incidence between the wrongly diagnosed and the genuine cases of scarlet fever, the former being the higher. It is of course impossible to examine this point directly for the whole of London, but I have examined the records for three years at my own hospital and failed to find this explanation true. During 1920, 1921 and 1922, 218 males and 244 females were admitted wrongly certified as scarlet fever. The mean ages were, males 8.32, females 8.83; both lower than the mean ages of cases notified in 1920.

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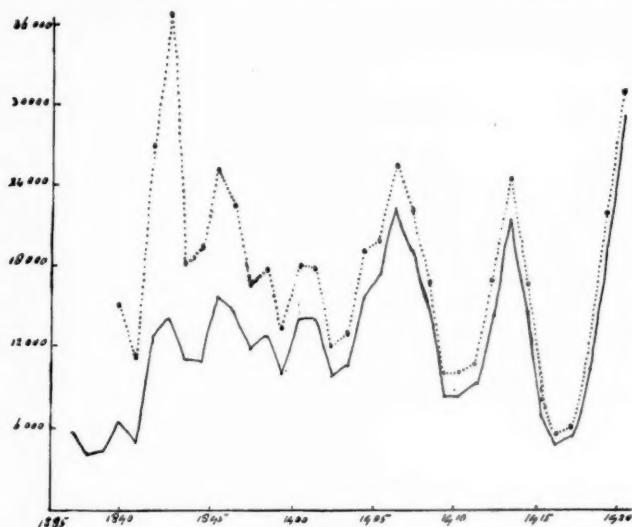


Chart I  
Scarlet Fever in London.  
Cases notified .....  
Cases admitted to hospital. —

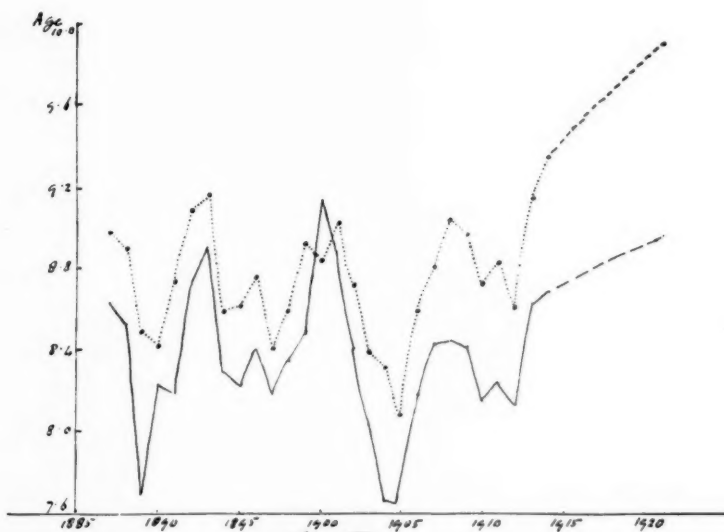


Chart II  
Scarlet Fever in London. Mean Age of cases admitted —  
Mean Age of cases notified .....  
Males —  
Females .....

## (II) PROPORTION OF THE TWO SEXES.

The number of notifications is always greater for females than for males, in the totals for the whole series in the ratio of 1 : 1'133. In each year also the females admitted outnumbered the males.

The variations here also seem beyond the range of chance variations, but the evidence is not very strong. For the year 1917 the P.E. is  $\pm 0'031$  and for 1914  $\pm 0'010$ . Of the twenty-six years shown in the first half of the table the ratio is in seven instances between  $1'133 \pm 0'020$ . There is a slight tendency for the excess of females to increase towards the end of the period.

## (III) THE GENERAL SHAPE OF THE CURVES.

If any one year be plotted out with age as abscissa and number of cases at that age as ordinate we get a very skew curve. The mode or highest ordinate is usually at age 4-5 years; though as the years 5-10 are all grouped together it is possible that the maximum really occurs after 5. To examine the similarity or differences between the curves for different years we can employ three methods: (1) To plot all the curves on one sheet of paper. (2) To reduce all the figures by proportion to a common denominator. (3) To fit curves to each and examine the results and compare the differences to the probable error.

(1) By plotting two years' figures together we at once see the close correspondence in shape. But when the numbers concerned vary widely it is necessary first to reduce them to a common denominator.

This is the first method I adopted in examining my material. I took the figures, using both sexes combined, and reduced the yearly admissions from 1887 to 1914 to a common denominator 1,000. I soon found that it would not be possible to plot all twenty-eight curves on one sheet of paper without inextricable confusion. Even five years plotted together gives curves too close together to be distinct.<sup>1</sup>

(2) If, however, we reduce all the figures in proportion to 1,000 cases and examine them side by side certain facts can be observed (*see* Table IV). The proportion of cases of any one age period varies widely. Thus to take for instance the cases between 5 and 10 years old. In 1889 these were  $440'4$  per 1,000 =  $44'04$  per cent. From 1887 to 1896 inclusive the figure is always above 400. In the next seven years it is six times below 400 and once above. After 1903 it is again always above 400. The extreme limits are  $440'4$  in 1889 and  $375'7$  in 1901. The probable errors of these figures are  $3'4$  and  $5'0$  respectively, so that here again the divergence is much larger than chance variations can account for. Taking the whole series, the frequency of cases between 1 and 5 years is exceptionally low in 1892 but high in 1903 and 1904, and again low in 1913 and 1914. The age-period 5-10 has been described above; 10-15 reaches a maximum in 1892 and again in 1914, and a minimum in 1904; 15-20 and 20-25 both show maxima at 1901 and minima at 1889 and 1905 with a broader depression from 1894 to 1898; from 25-30 the figures are nearly similar, a maximum at 1901 but minima at 1891 and 1903. We thus see that there must be special influences that alter the age distribution in scarlet fever from year to year and that these are not quite irregular. In some of the cases there is a hint of a seven-year period of fluctuation, but not definite.

<sup>1</sup> If, however, we choose two years with a wide difference in mean age, the two curves become sufficiently distinct.

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(3) The fitting of a mathematical curve. I have not yet succeeded in this method. I have calculated the moments in nineteen cases and attempted to fit curves of Pearson's types. In all cases the curve that gives the closest fit to the data taken as a whole shows complete want of correspondence at the first five years of life, where our data are given in greatest detail. I conclude that these curves will not fit our data.

TABLE IV.—SCARLET FEVER. CASES ADMITTED TO METROPOLITAN ASYLUMS BOARD HOSPITALS. BOTH SEXES. AGE-DISTRIBUTION PER 1,000 PERSONS.

Year														
Age	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900
0-1		9.5	7.3	7.6	9.7	9.3	8.2	10.5	10.7	8.8	11.8	9.7	8.6	7.9
1-		34.8	29.9	31.4	31.2	*28.2	32.4	36.9	39.4	33.8	37.9	33.7	31.6	32.0
2-	279.7	60.7	73.4	66.8	65.4	*53.2	57.4	64.5	67.1	64.8	73.7	61.0	64.1	65.3
3-		91.6	103.5	94.0	89.0	*81.1	81.3	92.5	91.5	91.0	98.2	96.7	95.6	90.2
4-		104.3	105.0	106.2	98.7	*95.2	96.7	*95.2	99.1	95.9	105.3	112.0	108.5	99.9
5-10	419.9	414.5	440.4	432.0	428.3	417.8	*403.4	421.1	407.5	408.8	399.1	408.0	393.0	386.8
10-	164.0	163.3	148.7	161.5	169.9	197.6	189.9	177.2	184.6	194.2	174.1	172.2	178.2	182.6
15-	70.3	61.7	*48.1	51.7	59.8	63.7	68.5	54.1	54.2	53.3	53.7	55.4	62.4	70.1
20-	133.4	30.4	*21.7	27.4	27.5	27.6	31.2	*22.8	22.9	24.6	22.6	24.8	28.6	35.1
25-	115.2	13.6	11.5	11.2	*9.5	13.4	15.1	13.6	12.2	13.4	11.4	12.6	15.5	15.9
30-	18.5	8.4	7.3	*5.5	6.8	6.9	18.7	6.6	5.6	6.1	6.8	17.8	7.6	7.7
35-	14.4	4.3	*2.0	2.1	2.8	3.0	3.6	*1.9	3.4	3.5	2.5	2.9	4.0	4.3
40-	2.4	1.6	1.1	0.8	1.0	1.8	1.6	1.6	1.1	1.5	1.5	1.2	1.3	1.5
45-	1.2	0.9	0.4	0.9	0.4	0.4	0.8	0.5	0.5	0.4	0.8	1.0	0.5	0.5
50-	1.0	0.9	0	0.2	0	0.2	0.5	0.4	0.4	0.5	0.4	0.2	0.2	0.2
55-	0	0	0	0	0	0.2	0.2	0.1	0.2	0.1	0	0.2	0	0.1
60-	0	0	0	0.3	0.2	0.2	0	0.1	0	0	0.1	0.1	0	0.1

Age	1901	1902	1903	1904	1905	1906	1907	1908	1909	1910	1911	1912	1913	1914
0-1	10.6	11.0	9.7	9.5	10.2	8.6	8.8	8.2	8.6	6.9	*5.4	7.7	6.3	7.9
1-	37.2	38.8	48.4	41.3	41.0	37.0	*30.8	33.4	37.9	34.8	33.9	35.4	*28.8	31.8
2-	70.6	72.9	*80.2	78.1	75.3	71.0	*61.5	62.4	69.7	68.3	66.6	67.4	57.4	*55.4
3-	89.0	99.2	102.5	106.6	102.9	96.2	87.1	*85.1	86.0	100.0	84.6	93.9	79.1	73.8
4-	102.8	108.6	114.9	122.1	121.0	115.3	104.3	105.9	*100.7	109.1	110.1	108.0	91.7	96.7
5-10	*375.7	390.1	388.4	413.8	423.5	409.3	427.0	419.6	413.9	429.9	427.1	427.1	435.5	421.9
10-	169.5	156.2	145.7	*130.7	142.0	159.8	172.5	173.7	171.9	*148.1	168.6	162.3	191.5	194.6
15-	173.5	60.1	51.8	43.1	*39.2	46.8	49.1	45.0	48.6	42.9	44.1	46.2	51.3	55.2
20-	139.6	31.5	29.4	28.3	*21.6	26.2	22.2	28.2	26.7	24.7	24.4	21.7	24.7	26.5
25-	118.3	16.5	*13.1	14.2	13.5	14.8	16.3	15.7	18.7	18.8	19.6	14.8	17.3	17.9
30-	7.1	19.3	8.6	7.5	*5.9	8.8	7.4	8.7	8.8	10.5	9.6	10.4	8.5	10.4
35-	3.2	3.2	4.1	3.7	2.5	3.2	3.6	4.5	5.4	4.6	3.0	3.5	3.7	4.8
40-	1.4	1.4	1.2	0.8	1.2	1.3	1.4	2.5	1.8	1.6	2.3	1.4	2.2	2.0
45-	0.5	0.9	0.4	0.4	0.5	0.7	0.4	1.2	0.9	1.0	0.7	0.4	1.1	0.6
50-	0.3	0.3	0.3	0.2	0.2	0.1	0.3	0.5	0.6	0.2	0.1	0.1	0.4	0.2
55-	0	0.1	0.1	0	0.1	0.1	0.1	0	0	0.2	0.1	0.3	0.1	0.2
60-	0.1	0	0.3	0	0	0.2	0	0.3	0.1	0.1	0	0	0.1	0.1

\* Minimum year. † Maximum year for this age-group.

A problem which has more interest than most of the other features of a curve is the age of maximum frequency or mode. If the data were given for several years between 0 and 10 this would show in the data themselves. But as the mode falls near the age of 5, where our data suddenly change from a one to a five-year period it does not show. If a suitable curve were found to fit the data we could calculate the mode.

## Conclusions.

(1) Curves showing the age-incidence of scarlet fever in London and elsewhere have been published in great number. These curves resemble each other very closely, so much so that one's first impression is that the differences are due to chance. However on calculation these differences, though small, are greater than can be explained thus.

(2) The mean age in the two sexes differs by from  $\frac{1}{2}$  to 1 year or more. The difference tends to increase in the later years.

(3) Mean age of cases notified is higher than that of cases admitted. The cause of this difference is obscure.

(4) Comparing mean age of cases in each year with prevalence of scarlet fever, there is a strong tendency for both to rise together in seven-year periods. Thus, the mean age shows maxima in 1887, 1893, 1900, 1908 or 1909, 1914 and 1921. Maximum prevalence occurred in 1887, 1893, 1907, 1914 and 1921. On the other hand, a minimum prevalence occurred in 1900. Probably it is chiefly owing to this that no correlation can be shown from the figures available.

*Postscript.*—Since writing the above paper certain fresh material has become available. From Manchester I have received by the courtesy of the Medical Officer of Health a complete series of age tables for the past thirty-one years, over 73,000 cases. The two sexes are not separated, and the age-periods used differ somewhat from those in the London tables. The first ten years are given separately instead of the first five; but at the other end ages are more compressed; ages over 25 are given in ten-year and not in five-year periods. Also the London figures for 1921 are now published in the usual form, except that the first ten years of life are given separately. Both these

TABLE V.—SCARLET FEVER, MANCHESTER. BOTH SEXES.

Year	Cases notified	Mean age*	Year	Cases notified	Mean age*
1891	1,138	7.742	1907	2,732	8.807
2	1,671	7.949	8	2,893	8.855
3	2,031	8.487	9	3,700	8.916
4	2,230	8.004	1910	2,324	8.290
5	2,302	7.596	1	1,939	8.225
6	2,389	8.122	2	1,840	8.323
7	1,790	8.237	3	3,715	8.795
8	897	7.812	4	4,712	9.892
9	1,467	8.734	5	2,922	9.856
1900	2,507	8.713	6	1,185	9.432
1	2,692	8.988	7	829	9.050
2	2,282	8.330	8	779	9.612
3	2,012	8.418	9	1,758	9.727
4	2,063	8.107	1920	3,829	10.345
5	1,975	8.561	1	5,400	10.873
6	3,075	8.975			

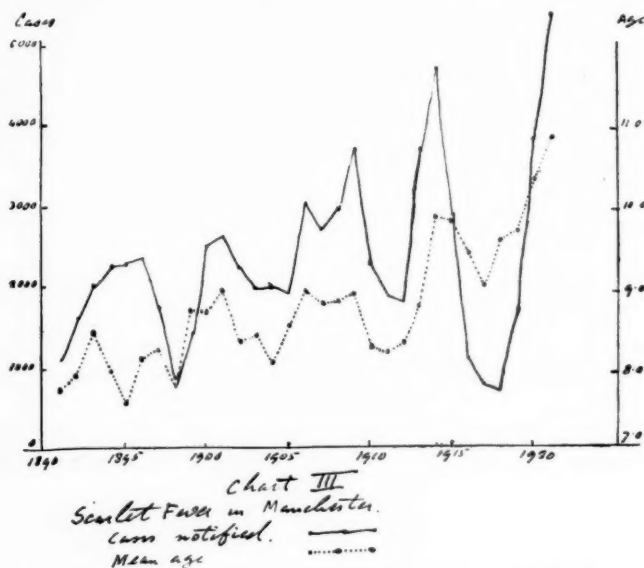
\* The mean ages in this column must not be compared directly with the corresponding years for London cases (Table III). First, because there the sexes are given separately. Secondly, the grouping together of ages from 5-10 in the London table raises the mean age. The London figures for 1921 now available can be calculated by both methods.

			Mean age	
			Males	Females
Using the 5-10 years group	...	...	9.106	10.050
Using those years separated	...	...	9.017	10.006
Difference	...	...	0.092	0.044



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series throw new light on the modal age, or age of greatest prevalence. In London, for 1921, both sexes show a maximum prevalence between 5 and 6, not between 4 and 5 as previously supposed. In Manchester taking the thirty-one years together there is also a definite maximum at 5-6. If, however, we examine each year separately there is a definite increase as time goes on. In the first eleven years the maximum occurs seven times between 4 and 5 and four times between 5 and 6. In the remaining twenty years the maximum occurs fifteen times between 5 and 6, four times between 6 and 7 and once between 7 and 8. In other respects the figures for Manchester afford good confirmation of the conclusions already arrived at from the London figures. For prevalence the two series correspond closely from 1914 to 1921. In both towns there were well-marked maxima in the two named years and there



was a very pronounced minimum in 1917 or 1918. For the remainder the correspondence is not close. Manchester also shows an attempt at a seven-year period. Several of the maxima are flat-topped and are irregular, but the chart shows five well-marked minima, all at exact seven-year intervals. For mean age also the Manchester chart shows, at least as clearly as the London chart, a tendency for high mean age to occur in years of high prevalence; also a steady rise in mean age of about two years.

### DISCUSSION.

Dr. (now Sir WILLIAM) HAMER said that in Dr. Turner's Table III (p. 21) there was, as he said, the striking anomaly that in 1900 the mean age of males exceeded that of females, while in all other years the mean age of females was the greater. Mr. B. E. Spear had however informed him that in that year an outbreak affecting

twenty-one constables (of average age 25 years) occurred, and so the exception might here, at any rate, be regarded as proving the rule. Dr. Turner's fourth conclusion, that there was a rise in mean age at times of epidemic prevalence of scarlet fever was particularly interesting. This result tallied with conclusions, submitted to the Epidemiological Society in 1896,<sup>1</sup> based upon mortality statistics. The charts accompanying the paper of that year showed: (1) That in small-pox, while age-periods 0-1 and 1-2 suffered lightly at times of epidemic prevalence, the age periods 2-10 and (still more notably) 5-10 suffered distinctly at such times. If the deaths at 0-2 were taken as 100, 2-5 and 5-10 (prior to 1870) rose in epidemic years, but later (after 1870) 10-25 suffered heavily and took up the role of presenting exacerbations in epidemic years. (2) In scarlet-fever, age-periods 0-1, 1-2 and 2-3 suffered lightly, 4-10 heavily, during epidemic years. If deaths 0-2 were taken as 100, the swing of the 4-10 waves clearly stamped its impress upon the periodicity of all ages. Sir Shirley Murphy, in 1898, examined the seasonal (minor) waves, and showed that, in both scarlet fever and diphtheria, there was an excessive incidence upon school ages in the autumn, but that infants were attacked in marked excess during August, when the curve for all ages and especially for school ages showed a depression.

Question necessarily arose as to the significance of these phenomena. The early writers on the epidemic wave stressed variation in the germ, though Netten Radcliffe was always impressed with the influence exerted by accumulations of susceptible persons. A. Ransome, in 1880 and in 1882, pointedly drew attention to the latter influence and thus, just at the commencement of the bacteriological era, an epidemiologist emphasized the influence of soil as well as of seed, and hence the localist as opposed to the contagionist point of view. Ransome seems to have concluded, however, that a simple "age theory" as he termed it, would not by itself suffice. Consideration of the charts exhibited in 1896 convinced him (Dr. Hamer) that the simple "age theory" might be helped by the assumption that there was special liability to attack at particular ages, e.g., at school age. This view of the question was later generally confirmed, though in some respects modified as to details, on study of the measles wave and of the distractingly complex influenza wave.

In measles the germ was remarkably stable and a mere mechanical explanation was almost sufficient. In influenza, on the other hand, there were reasons for thinking that the organism itself might be subject to mutation. Scarlet fever lay betwixt and between, and here it was clear that some environmental influence susceptible to changes in temperature and humidity was at work. In 1917 he (Dr. Hamer) had set out a summary of reasons for holding that this influence was the common flea. The age-incidence phenomena themselves might also perhaps be adduced as giving some further support to a flea hypothesis. These phenomena might be regarded, in Sydenham's phrase, as an instance of "Nature playing tricks"; but really, as was the case with Sydenham's instances, they supplied valuable clues which afforded help in threading the way through the labyrinth of epidemiological difficulties.

Dr. ISSERLIS said that while it was doubtful whether all the variations referred to were significant, the author had certainly made out a case for the continued publication of the scarlet fever tables. As regards the difficulty found by Dr. Turner in fitting frequency curves to the data it was not to be expected that curves based on material grouped in five-year periods should give a good fit to detailed frequencies in the separate years of the first period. In Table III, for example, the mean age for admitted males was given correctly at 8'844 years and was based on separate figures for the first five years. But in fitting a curve to the grouped frequencies the mean would have to be taken at 8'618. He (Dr. Isserlis) said that Dr. Turner was impressed by the variability of the mean ages in successive years. For notified males the deviation of the mean of the sample from the general mean exceeded twice the skew deviation eight times, while the probability of such an excess for samples of constant size was only 0'0456, i.e., an event happened eight times in twenty-six trials which one would expect to happen only once in twenty-two trials. There was a danger however in this comparison. The

<sup>1</sup> *Trans. Epid. Soc. Lond.* (1896-97), 1897, xvi, p. 64.

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problem should really be attacked by the methods used by Mr. Soper in studying the frequency distribution of partitioning a population into a stated number of categories of variable content. Dr. Isserlis thought no conclusion could be drawn from the small correlations of  $0.12 \pm 0.13$  found between mean ages and numbers notified, apart from the fact of all the variations being in one direction. To a layman it seemed natural that a big epidemic should involve higher mean age in the same way that a larger army meant a higher mean age of soldiers during the war.

Dr. GREENWOOD suggested that the small arithmetical value of the correlation between mean age and prevalence might be a consequence of non-linearity of regression, that the correlation ratio might be larger and significant. Were this the case, the statistical result would come into line with what, as Dr. Isserlis had said, seemed *a priori* reasonable.

Dr. CARNWATH said that he would refer to one point only in Dr. Turner's paper, viz., the alteration in the age-incidence of the disease during times of epidemic. In periods of "normal" prevalence the disease affected mainly the early years of school life. He thought it was a natural presumption that when the wave rose the overflow should be forwards in the direction of the adjacent school years rather than backwards towards the years spent in the home. In the schools the children were aggregated in a way that made extension of an epidemic easy as compared with the opportunities for infection in isolated homes. He was disposed to think that some such explanation might also account for the gradual rise in the average age of incidence during the last twenty years. Home conditions were admittedly still far from satisfactory; nevertheless a gradual and substantial improvement had taken place; there was less overcrowding, and there was a higher standard of cleanliness, and families were of smaller size than formerly. The opportunities for infection, therefore, during the early years of life in the home had been considerably reduced, and as a consequence the natural tendency was towards a higher proportionate incidence on the later school years.

Dr. TURNER (in reply) alluded to one point not mentioned in his paper. If a child caught scarlet fever, he was necessarily nursed at first by his mother, and sometimes by the elder daughter. This might account for some of the excess of females over males at ages from 15 to 30.

### The Relationship between Rainfall and Scarlet Fever.

By JOHN BROWNLEE, M.D., D.Sc.

(Director of Statistics, Medical Research Council.)

IN April 1880, the late Dr. Longstaff<sup>1</sup> read a paper before this Society on the relationship between scarlatina, erysipelas, puerperal fever, &c., and showed that between certain of these diseases there was similarity in the amount in which they were present from year to year. The part of his paper, however, which concerns us to-night is that in which he showed that from 1855 to 1875 a close relationship appeared to exist between the amount of rainfall and the prevalence of scarlet fever, wet years being associated with less scarlet fever and dry years with more. From 1875 to 1880 the association was less marked. There is no doubt of this agreement, the only question is, as to whether it is accidental or permanent. The data used by Dr. Longstaff related solely to deaths. If the death-rate is high, this is no drawback, but from about 1874

<sup>1</sup> *Trans. Epid. Soc. Lond.* (1880-81), 1881, iv, p. 421.

onwards scarlet fever became a much less fatal disease and from the middle eighties the number of deaths have been relatively so small that it is hardly possible to trace accurately the epidemic variations of the disease from deaths alone. From this cause, therefore, the later figures in Dr. Longstaff's table do not weigh much negatively. The general correspondence between the variations of the weather and of scarlet fever was so marked however, that it seemed impossible to deny the association.

This conclusion I had accepted until I began to investigate the periodicity of epidemic disease. After examining the statistics of scarlet fever in a large number of towns it became evident that, as with measles and whooping-cough, the intervals between epidemics varied greatly in different places. If, therefore, there was an association between rainfall and scarlet fever, there must be local variations in the rainfall, of temporary if not permanent periodicity,

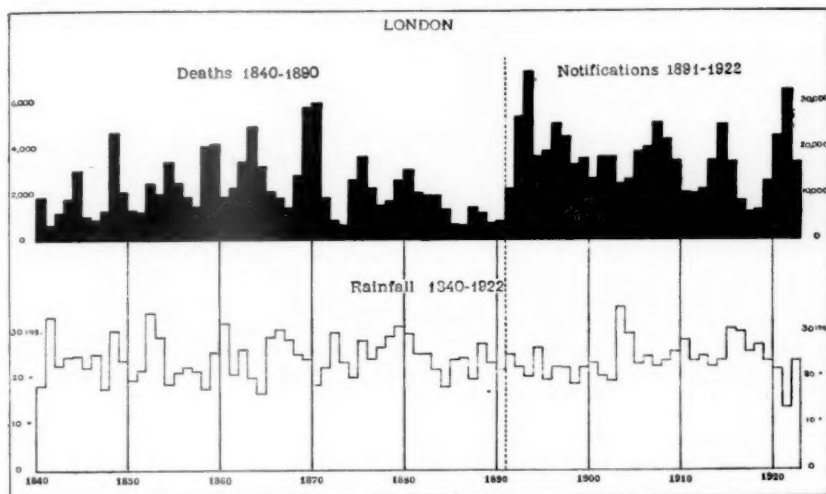


DIAGRAM I.—In this diagram the annual number of deaths from scarlet fever is shown from 1840-1890 and the number of notifications from 1891-1922 on a different scale. The course of the rainfall is graphed below.

in different districts of England and Scotland. This promised to open up not only a fresh chapter in epidemiology but also another chapter in meteorology. It seemed exceedingly unlikely that such variations could take place. It was determined, therefore, to investigate systematically a number of the large towns for which rainfall statistics were obtainable and for which the number of deaths or notifications were large enough to render the course of the epidemics of sufficient certainty to act as a basis for quantitative work. In England the towns selected were London, Birmingham, Manchester, Liverpool, Newcastle-on-Tyne; and in Scotland, Glasgow and Edinburgh.

In the first place it may be noted that the statistics of Manchester and Edinburgh show very little evidence of definite periodicity of scarlet fever. With regard to Manchester this is the third disease for which this has been found, measles and whooping-cough being the other two.

The first town to be examined is London. The facts are shown in Diagram I (p. 31). The amount of rainfall and the number of deaths from scarlet fever are graphed from 1840-90. From 1890 the number of deaths is replaced by the number of notifications. In the first part of this period from 1840-57, diphtheria is not separated from scarlet fever, but the amount of diphtheria at that time was very small, and checking the figures against such diseases as laryngitis and croup nothing but a small error can have been introduced. It will be observed that during the first period 1840-57, the correspondence between the amount of scarlet fever and the rainfall described by Dr. Longstaff is not present. From 1855-76, the association is very close. The correlation is high, being in the region of 0.7. From 1876-90, the correspondence again ceases. In the early period, intervals between epidemic waves of scarlet fever are very nearly five years but in 1890 a new epoch begins. The very large epidemic of 1893 is the starting point of a seven-years period, the succeeding epidemics culminating in the years 1907, 1914 and 1921, the epidemic due in 1900 not appearing. This fact will be referred to later. In this period it may be said that there is a fair correspondence between drought and scarlet fever, the very dry year of 1921 associated with the largest epidemic since 1893 serving to bring the subject again into public notice. It is, however, to be noted that the epidemic due in 1900 did not appear, although the years around this were associated with comparative drought, and that therefore it cannot be said that drought in itself will bring about an epidemic, if other conditions tending to prevent an epidemic exist.

Considering Glasgow next, we find that from 1855 to 1880 the period between the epidemics is very closely five and a half years. Here, again, some relation between dry years and scarlet fever can be made out. There are, however, some curious exceptions. The year 1857, a middle year in an epidemic wave, was a dry year in Glasgow and yet the autumnal outbreak of scarlet fever was absent, though marked in 1858, a year considerably wetter though not a wet year. In Diagram II the average relationship is shown. This is obtained by taking the average number of deaths in each six months and the rainfall in each six months, choosing five and a half years' interval as the most constant phenomenon and adding together the corresponding numbers in each five and a half years' period. The inverse relationship is quite marked, but in view of the preceding remarks too much should not be made of it.

Notification was introduced into Glasgow in 1891. As in London, there was a great epidemic in 1893, but this was not the starting point of a seven-yearly period. The epidemic occurring in 1894 seems to have been the starting point of a strict five-yearly period; the acme of the next epidemic occurs in 1899; the epidemic due in 1904 does not appear. It was previously noted that in London, a like phenomenon occurred at the same time; the epidemic in 1907 turns up true to time as likewise do those in 1914 and 1919. It is to be noted that the year 1921, a year of large amount of scarlet fever in London, and a dry year both in London and Glasgow, was not marked by any epidemic of scarlet fever in Glasgow; the crest of the wave being passed, dry weather had not sufficient power of itself to sustain the prevalence.

Liverpool exhibited a very remarkable series of epidemics. In view of the importance of the subject, the Registrar-General gave me facilities for having the weekly deaths from scarlet fever in Liverpool extracted from the registers in Somerset House from 1853 onwards. One of the most regular periods with which I am acquainted is found to exist at this time, the period being exactly four years. In the accompanying diagram (Diagram III) the quarterly

deaths from scarlet fever have been graphed from 1857 to 1876. The rainfall in each year is indicated by circles below the graph; a dry year is shown by a circular black dot and a wet year by a circle enclosing a cross. A dry year is defined as one in which the rainfall is 3 in. below the mean, and a wet year one in which the rainfall is 3 in. above it. Years with rainfall between these limits are not marked; years for which the statistics of rainfall are not recorded are marked with a horizontal

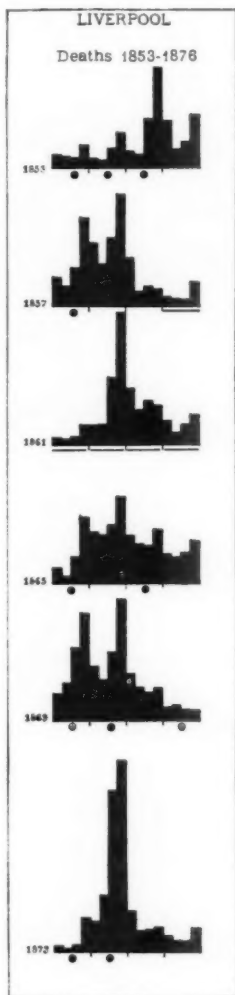


DIAGRAM III.

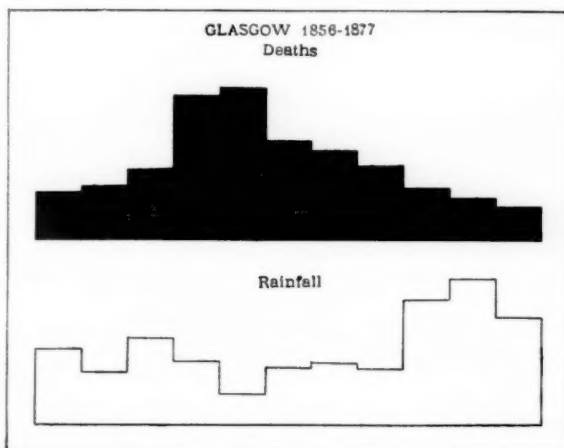


DIAGRAM II.—This diagram shows for twenty-two years, 1856-1877, the average number of deaths from scarlet fever in five-and-a-half-yearly periods and the corresponding average rainfall.

DIAGRAM III.—In this diagram the quarterly deaths from scarlet fever are shown in four-yearly periods from 1853-1876. A dry year, that is one in which the rainfall is less than the average by 3 in., is indicated by a black circle, and a wet year, one in which the rainfall is more than 3 in. above the average, by a circle enclosing a cross. For the years with a horizontal line beneath them, the rainfall records are not available.

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line. Here it is to be noted that on two occasions, 1858 and 1866, the largest unit of the epidemic wave occurred with a mean rainfall. In 1866 the amount of scarlet fever was comparatively small and the rainfall just fails to qualify that year as a wet year. Nothing definite can be said about 1862, for the rainfall is not recorded in Liverpool for that year, but in Manchester the rainfall is nearly exactly the mean. The year 1874 was remarkable for its dryness and the size of the epidemic was greater than on any other occasion. Subsidiary waves occur three times. Two of these, the years 1855 and 1857, were associated with dryness. The larger, however, occurred in 1869, which was a wet year.

With regard to the other towns it is not necessary to particularize especially. The same features are apparent as have just been described. One remark, however, may be made about Edinburgh. In this city there has been no regular periodicity of the disease, so that it might be possible to find a closer association between the rainfall and scarlet fever. The association found is not remarkable.

As to the relationship, then, between rainfall and scarlet fever, I think it must be said that it is not causal. There is, a strong presumption that if the conditions are suitable for an epidemic, a dry year will tend to increase its size and a wet year to diminish it. As, however, at the present moment there is apparently no satisfactory statistical method of dealing with an association such as this; it cannot be measured quantitatively.

#### DISCUSSION.

Sir GEORGE BUCHANAN thought that this investigation was very useful. It gave them facts by which to check the different statements which were often made about the relation between scarlet fever and wet seasons. It would be interesting to know whether the comparison of scarlet fever epidemic years and years with high rainfall had also been extended to months. With such a variable climate as that of the British Isles the total annual rainfall might be quite misleading, for example, as an index of the rainfall in the autumn. In this connexion he thought another question deserved study, namely the facts about seasonal incidence of scarlet fever in different countries and their explanation. Speaking from memory he believed that the characteristic autumn maximum of Great Britain was not found in some Continental countries and was sometimes replaced by a spring maximum. In North America and Japan the maximum incidence of scarlet fever undoubtedly fell in the early months of the year.

Dr. BROWNLEE (in reply) said that when the correspondence between the amount of scarlet fever and the rainfall was examined in smaller periods than the year the relationship became less definite.



## Section of Epidemiology and State Medicine.

President—Dr. RICHARD J. REECE, C.B.

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### Progress and Problems in Epidemiology :

#### PRESIDENT'S ADDRESS.

By RICHARD J. REECE, C.B., M.D.

#### INTRODUCTION.

MY first duty is to thank the Section of Epidemiology and State Medicine of the Royal Society of Medicine for the very great honour conferred on me by the members in electing me to the important position of President. It is an honour which I deeply appreciate, but I hesitated to accept it, for while I can admit that for many years I have been a student in the science of epidemiology (I have been a member of the Epidemiological Society since 1892), I realized my limitations and the difficulties of worthily upholding the position which so many distinguished men have filled. In accepting the Presidency I did so with the belief that in electing me to the office you intended the compliment to be paid to the public health department of the State, which has furnished so many members to this Society, in which I have the honour to serve, and which in the changing circumstances of the times is now styled the Ministry of Health.

My second duty is to offer my apologies to the Section for the delay that has occurred in the delivery of the customary Presidential address. In the first place, this was due to my leaving England on an official visit to the United States of America, and secondly, to the fact that on my return to this country an outbreak of small-pox kept me more than busy. The time at my disposal for preparing a presidential address has been so limited that I could not hope, had I the ability, to place before you a reasoned statement of the present prevailing opinions in regard to epidemiological research and I can only crave your kind indulgence.

#### THE HISTORY AND AIMS OF THE OLD EPIDEMIOLOGICAL SOCIETY.

The Commemoration Volume of the Epidemiological Society of London, issued in 1902, in celebration of the Jubilee Anniversary of the Society, records that the Society owed its origin to Mr. J. H. Tucker, of Berners Street, who, under the *nom de plume* of "Pater," advocated its formation in letters which he published in the *Lancet* in 1849. It was suggested that the first work of such a Society would be to deal with cholera, but later the scope of its influence was extended to other diseases. The ultimate result of these letters in the *Lancet* was a public meeting which was held on July 30, 1850, under the presidency of Lord Ashley, afterwards the Earl of Shaftesbury. The foundation of the Society followed and Mr. Tucker was one of its first three

secretaries. The first meeting took place on December 2, 1850, and Dr. Guy Babington, F.R.S., the first President, when delivering the opening address defined the object of the Society in these words:—

"To endeavour by the light of modern science to review all those causes which result in the manifestation and spread of epidemic diseases—to discover causes at present unknown, and investigate those which are ill understood; to collect together facts upon which scientific researches may be securely based; to remove errors which impede their progress; and thus, as far as we are able, having made ourselves thoroughly acquainted with the strongholds of our enemies and their modes of attack, to suggest those means by which their invasion may either be prevented; or if, in spite of our existence, they may have broken in upon us, to seek how they may be most effectually combated and expelled." (*Lancet*, December 7, 1850.)

In 1913, when the title of the Section was changed, the objects of the Section were defined as follows: "For the investigation of epidemic and endemic diseases in respect of the circumstances and conditions which favour, prevent, or are otherwise associated with their occurrence, their persistence or their variations in type or character."<sup>1</sup>

To what extent and with what success the Epidemiological Society has contributed to the science of epidemiology and to medical literature can be realized by reference to the index of the papers that have been read at its meetings, and to the reports of its various committees which have been formed from time to time for the purpose of studying special branches of epidemiology; and certain of these reports have been printed by order of both Houses of Parliament. Though much has been accomplished there is yet much to be learnt and there are many problems to be solved; the work for which the Society was founded remains, and added knowledge has demonstrated the vast field and complexity of the problems that have yet to be unravelled.

From the report in the *Lancet* of August 3, 1850, on the meeting which led to its foundation, we learn that the objects of the proposed Society were two-fold. One is set forth in the words I have quoted from the address of the first President, and the other is "To appeal to the public for help and encouragement in effecting a mighty effort for the public weal." It is clear that in the early days of the Society contributions to its funds were expected, and in the laws of the Society defining the duties of the Treasurer and of the Secretaries it is stated that these last-named officers "shall at each meeting . . . announce donations."

Laymen were solicited to join our body as donors and as annual subscribers; it was maintained that we ought to be subsidized by Government itself and a contribution was actually solicited by a petition to the Prime Minister. The early attempts to induce lay members to make common cause with us signally failed, and the first President, Babington, who had held office from the commencement of the Society in 1850, in his farewell address in 1864, had no hesitation in stating that "we must support our character as scientific investigators, apart from all considerations of popular sympathy, or we shall lose our dignity and self-respect, and, together with these, the influence and utility of our position and authority." He stated that if the Society had not done all the works which had been planned for execution at its formation, this was not to be attributed to want of zeal or inclination on the part of its members, but to the very restricted amount of funds at their disposal to carry out operations so extensive and so costly.

<sup>1</sup> *Proceedings*, 1913, vi (Sect. Epid. and State Medicine), p. 119.

While the Epidemiological Society's *Transactions* bear witness to the wide range of subjects considered at its meetings and to the merit of the contributions furnished by its members to the study of epidemiology, they also testify that many of the most important papers have been written by medical officers of the Royal Navy, the Army and the Colonial Services, and by medical men who have not served in any of the State medical services. Our first president, a pioneer worker of his time in many branches of medicine, was a physician to Guy's Hospital. He exercised a wide and beneficent influence over our Society during the first fifteen years of its existence. Although he had acquired experience abroad as a midshipman in the Royal Navy and in the Civil Service of the Honourable East India Company before he commenced the study of medicine, he never served in any medical department of the State. Without in any way detracting from the value or minimizing the high esteem that is due to the work of these men, it can be said that the elucidation of epidemiological problems has been closely associated with the public health department of the Civil Service.

The birth of the Epidemiological Society in 1850 took place at a time when John Simon had commenced to write, and the medical staff of the Privy Council came into existence. To this staff belonged Netten Radcliffe, George Buchanan, and others, the value of whose epidemiological work cannot be over-rated. When the State Medical Department was transferred to the newly constituted Local Government Board in 1871, other men joined the service, and the study of epidemiology was continued by Power, Thorne-Thorne, Ballard, Barry, Parsons, T. W. Thompson, Theodore Thomson, Bulstrode, and many more whose names are household words in the Epidemiological Society. Only last year we lost one of our members, who joined the Society in 1887, my old colleague Robert Bruce Low, whose industry, skill and perseverance materially assisted the advancement of our knowledge in regard to the prevalence and distribution of disease throughout the world. In a sense he continued the work which is specially associated with the name of Netten Radcliffe. While he held a strong view on many subjects, he had only one opinion, and that an honest one; and he was able to keep an unbiased mind for he loved truth for truth's own sake. His amiable disposition, courteous manner and his readiness at all times to place his knowledge and experience at the service of his medical brethren endeared him to all.

The Medical Department of the Local Government Board received a small annual sum from State funds, which was known as the Scientific Grant, and the use made of this, and the value of the work it was possible to subsidize, are shown in the annual reports of the medical officers of the Board.

A small amount is still allowed to the Ministry of Health, and is available to meet current needs of its medical department, but the State has realized the importance of investigation of medical problems, and the formation in the year 1913 of the Medical Research Committee, now known as the Medical Research Council, has been the result. When the Epidemiological Society was first instituted it advocated adequate endowment for medical research. We cannot claim that such a proposition solely emanated from that society, as it has been advanced by others, but we can congratulate ourselves that with the advent of the Medical Research Council one of its primary objects has been fulfilled. The science of epidemiology is not limited to one country, it embraces the whole world; it is not confined to one race of man, nor indeed to mankind alone, it extends to

the animal and vegetable kingdoms. It is closely associated with geography and history, with the work of the physiologist, pathologist, bacteriologist and chemist, with the science of statistics, and with clinical medicine. Its interests are far reaching, and this Section, devoted to its study, can look forward with satisfaction to the progress our branch of medicine is bound to make as a result of the comprehensive work carried out under the auspices of the Medical Research Council.

#### DISEASES COMMUNICATED TO MAN BY INSECTS.

One cannot look back without realizing how many advances the science of epidemiology has made during the last fifty years. Advance has been made by leaps and bounds since Laveran, in 1880, demonstrated the animate nature of the malaria germ, and expounded his views in regard to protozoan infection, and since Manson, in 1878, wrote on the development of the *Filaria sanguinis hominis*. Manson became a member of the Epidemiological Society in 1900, and his membership in the Section of Epidemiology and State Medicine ceased with his death in April last year. During this period his pleasing personality, and his willingness at all times to help others, won for him the goodwill of its members, while his wisdom in debate compelled their respect. He was a firm believer in the influence of the Epidemiological Society in promoting the study of epidemiology, and his interest in it was second only to that which he took in the London School of Tropical Medicine, to which he devoted much of his time. The foundation of this School was the direct outcome of a lecture he delivered in 1898 at St. George's Hospital on the need for special study of tropical diseases, and from the date of its initiation it was his spirit that inspired the valuable work done at this School. Born in 1844, Manson was only 21 years old when he went to Formosa, and commenced the study of tropical diseases, and with a success that has made his name famous throughout the world.

It is difficult to forecast what will be the ultimate effect on epidemiology of the many contributions to medical and scientific knowledge furnished by Manson during the half-century of his active professional life.

In his Presidential Address, before the Epidemiological Society, in November, 1900, Sir Patrick Manson set forth what he thought should be the future lines of research in the elucidation of the many epidemiological problems brought prominently before the world, by the necessity for including the protozoa and nematodes among the important pathogenetic organisms affecting man, and by the knowledge that many diseases are spread in a variety of ways by blood-sucking arthropods. He considered that each problem, after thoughtful formulation, should be carefully worked out by a special investigation. He expressed the opinion that this was the only method of advance, as it was the only way in which to economize the somewhat limited supply of pathological and epidemiological energy in the market. He also suggested that as each investigation would probably extend over a considerable period—probably one or two years—in order to secure continuity of work and to minimize risk of interruption by ill-health, or other contingencies, it would be better to have two investigators for each subject. He concluded his address with these words:—

"It will be strange if in a country which provides liberally for what might be called sentimental expeditions—such as the discovery of the North Pole, or for settling mere geological points of not very great practical importance, such as the determining of the process by which the Coral Islands acquired their peculiar conformation—it will be

strange if in such a country funds cannot be provided to attempt, in the way I suggest, or otherwise, the settlement of important questions affecting the life and comfort of men and the material prosperity of the country."<sup>1</sup>

During the twenty-two years that have elapsed since Manson delivered the address, there has been considerable stimulus in the study of pathology and bacteriology, sciences that directly improve our knowledge of epidemic disease though not of the epidemicity of disease. Many workers have entered these fields of study, but it cannot be said that there has been any comparable influx of workers in the domains of epidemiology. There are few men who have the time and opportunity to devote to this science, and however rich the reward that may fall to the successful worker from the fact that he has materially assisted in advancing knowledge and in affording relief to suffering humanity, it is nevertheless a sad truism that, judged on the sordid basis of finance, there are other branches of medicine which are more remunerative. To be able to work men must be able to live, and to be able to live necessitates the possession of the essential means of supporting life.

Manson's early work on filariasis gave an enormous impetus to the study of the part that insect life plays in the transmission of disease to man, and has resulted in definitely linking up certain diseases with blood-sucking flies; malaria, yellow fever, sleeping sickness, dengue and the maggot disease of Trinidad and Central and South America are instances of this. It has been suggested by Sambon, colleague and friend of Manson's, that some blood-sucking fly, of cosmopolitan distribution, may be responsible for the spread of leprosy. Should this prove to be the case it will be interesting to know whether this fly is to be found in England at the present time. The absence of this fly would help to explain why cases of leprosy do not now occur in persons in this country unless they have been resident in places abroad where leprosy is endemic; while the presence of this fly would add another to the many problems for solution, namely, why the condition does not spread to our indigenous population from the lepers who have returned to this country from abroad.

#### DISEASES COMMUNICATED TO MAN BY ANIMALS.

If we turn for a moment to diseases that are spread from animals to man, we have an instance, and one we cannot often observe, of the effect of a legal enactment, within a short period of time from its being put into operation, having the direct result of eliminating a disease from our midst, and in regard to which the cause and effect can be seen distinctly. This is the Rabies (Muzzling of Dogs) Order of the Board of Agriculture and Fisheries which was first put into operation in 1889, and which was in force in one or another part of the country for fifteen years. As the result of its enforcement and through the restrictions placed on the introduction of dogs from abroad, hydrophobia ceased to exist in this country; and for a period of fifteen years we were free from the disease. Re-introduced during the war period in 1918 the infection speedily disappeared on the re-enactment of the Order, and the Order was withdrawn finally in 1922.

While it is generally accepted that the milk of cows suffering from tuberculous disease of the udder is capable of conveying tuberculosis to man, we observe an extraordinary attitude on the part, not only of the medical profession but also of the veterinary profession, namely, that of rejecting any suggestion

<sup>1</sup> *Trans. Epid. Soc. Lond.* (1900-01), 1901, xx, p. 10.

that the milk of cows can convey other disease conditions to man. An instance of this is the group of symptoms closely allied to those with which we are accustomed to associate with the malady known as scarlet fever, and which, from the fact that the inculpatated cows were at a farm at Hendon, became known as the Hendon cow disease. The reasoned plea advanced that the observed illness in man was the direct outcome of the consumption of the milk of the affected cows was combated with a strenuousness which was remarkable. The opposition to the theory that such a disease in man could be acquired through the consumption of specifically infected milk remains, notwithstanding that the application of the discovery of Horrocks, that Malta fever was spread by goats acting through their milk, had the immediate effect of eliminating a milk-borne disease which for many years had materially impaired the military efficiency of the British garrison at Malta. Like conditions to those described in the Hendon outbreak, in which the malady in the human species can only, in the light of our present knowledge, be referred to the consumption of milk, and in which the cows that supply the milk are found to be suffering from local disease of the udder and possibly from a general constitutional affection, continue to occur from time to time. There is a recent incident in which the removal of an affected cow from the milch herd of a particular dairy was followed by the cessation of illness among the consumers of the milk.

At the time that the theory of the Hendon cow disease was first advanced the objections raised may have been due in some measure to the fact that the bacteriologists of those days endeavoured to link up the observed phenomena with what was then considered the causal micro-organism of scarlet fever, and their failure may be wholly or in part responsible for the opposition directed against any possible association of the malady in man with cow disease. The amount of personal feeling that has been introduced into the controversy is regrettable. If the Hendon cow disease is a disease entity, from the point of view of a symptom group, abuse of the theory of its causation will not affect its validity, and I am tempted to quote a remark made a couple of years ago by a member of this Section that "we need all our wits for construction, and have none to spare for the manufacture of wounding sarcasms or the revelation of others' shortcomings."

Anthrax is another disease that is communicated directly or indirectly from infected animals to man, but in it the infection is limited to the individual and does not spread from him to the community. For many years it has been known that the infection can be acquired by man through handling specifically infected skins. An instance occurred during the war, in which Professor J. F. Tulloch, of Dundee, then a Lieutenant in the Royal Army Medical Corps, found the *Bacillus anthracis* in the cerebro-spinal fluid of a man who was thought to have died of cerebro-spinal fever, and on continuing his investigation he was able to demonstrate that the infection had been acquired through the respiratory tract. By so doing he added another link to the chain of evidence that anthrax is one of the diseases that is air-borne. A more recent instance in connexion with anthrax which is of interest is that of a man who died of anthrax, and who, immediately before his fatal illness, had been engaged in unloading bone meal from a vessel in one of the London Docks. Colonel P. G. Stock, who investigated the matter, was able to produce evidence that certain samples of the bone meal obtained from the cargo of the vessel were infected with anthrax, and the question at once arises whether the sporadic outbreaks of anthrax that occur among animals in this country are associated with the use of bone meal for manure.



Recent researches have demonstrated the part played by the rat in rat-bite fever, epidemic jaundice, trichinosis and septic pneumonia, whilst the known fact that the rat-flea is responsible for conveying the infection of plague has in great measure directed preventive measures against the rodent rather than against man, so far as the bubonic and perhaps the septicaemic form of the disease is concerned.

It is interesting to recall the views held during the last half-century in regard to the spread of plague.

The Commission of the French Academy of Medicine, in 1884, concluded that plague should be regarded as an endemic disease of Egypt occurring in communities living upon alluvial and marshy soils and on the banks of great rivers such as the Nile, and that it was associated with badly ventilated and over-crowded houses, great accumulations of putrefying animal and vegetable matters in the vicinity of dwellings, unwholesome and insufficient food, excessive physical and moral misery and the neglect of the laws of health in public and private life. It came as a surprise a few years later to find that plague had established itself in other parts of Africa and in Asia. The danger of infection was thought to be proportionate to the fouling of the atmosphere surrounding the patient by the effluvium from his body and breath, and that the infection was peculiarly easy of destruction by free dilution with air. It was recognized that the infection could be carried by fomites, such as clothing and bedding, and that the condition for infection of articles of clothing and bedding was their intimate use by, or association with, the sick. Direct contact with the sick was not considered essential to transference of infection. We can see now that the environment of plague-infected areas was such as to encourage infestation by rats and that the conditions in which infection was acquired were those that were favourable to its transmission by the rat-flea. As the result of researches into relationship of the flea to the rat, and of the rat to man, plague came to be regarded from the point of view of epidemiology as essentially a rat disease in which human beings may participate. The Advisory Committee for Plague Investigation in India concluded their interim report of 1910 with the statement that: "In districts which suffer annual epidemics of plague, the rat epidemic, on which the human epidemic depends, occurs during some part of that season when the prevalence of fleas is greatest." The seasonal prevalence of plague is therefore dependent on the life history of the flea. In the space of the last quarter of a century during which we have been adding to our knowledge of plague, we have seen this disease spread from place to place and we recognize that it has been established in districts far removed from Egypt and India, viz., among the ground squirrels of California and the garbilles and multimammate mice of South Africa.

During the war large stocks of merchandise, accumulated at certain ports abroad, waited for opportunity of shipment, and much of this afforded excellent opportunity for the harbourage of rats as well as ample food and facilities for nesting and breeding purposes: it is admittedly difficult to prevent rats being carried on board ship with cargo at such ports as Bombay. The exigencies of the situation prevented systematic, or indeed any, fumigation of ships for rat destruction and the rat population increased on board ships, and much dunnage accumulated on vessels. An instance of this, and probably an extreme case, is that of a vessel fumigated by burning sulphur in January, 1920, in the Port of London. After fumigation 1,466 rats were found dead and thirty-five loads of refuse were removed from her holds.

The requirements of the United States of America that all ships trading to



ports in the States shall be "de-rat-ized" every six months has given a stimulus to the use of hydrocyanic acid gas for the destruction of rats and rat-fleas on board ships, and evidence of the properties of this gas being capable of destroying human life have not been wanting. Nevertheless the advantages of this gas in freeing vessels and especially vessels of large tonnage, from rats, fleas and bugs are so great that systematic investigation in regard to its properties under circumstances met with on shipboard and varying conditions of temperature and mixture with other gases is now being undertaken abroad and in this country.

The systematic "de-rat-ization" of vessels which has been practised by many nations since the conclusion of the war has had a remarkable effect in lowering the rat population on board vessels. In our ports, and in the ports of the United States, very few rats are now found on vessels after they are fumigated. With the disappearance of rats from vessels the possibility of plague being ship-borne from port to port becomes remote. The difficulty of devising adequate measures on land to exterminate the infection in rodents and like animals remains.

#### "CARRIERS" OF DISEASE.

When it became known, primarily through the work of bacteriologists, that man, as a "carrier," played a part in the spread of infectious disease, a new problem in epidemiology presented itself and inspired fresh efforts to cope with it. Nevertheless a warning was forthcoming from the epidemiologist that the discovery should not be relied on as a final solution of disease causation. The carrier subject was eagerly investigated by the bacteriologist, experimental pathologist and public health administrator. Enteric fever, paratyphoid, cholera, dysentery, cerebro-spinal fever, poliomyelitis, diphtheria, pneumococcus pneumonia, influenza, were all subjected to close scrutiny with the object of obtaining effective control over these diseases. It became recognized that there were two types of carriers, the transitory carrier and the chronic carrier, and that the latter may be the subject of periodical recurrences during which he may be peculiarly liable to spread infection. It was thought that he was responsible for the persistence of infection in endemic form and for recrudescence of infection in epidemic or explosive form.

But we are far from understanding the part played by the diphtheria carrier, and while we know that a large number of diphtheria carriers exist, it is only occasionally that a carrier transmits the clinical disease to others. The test of virulence of the *Bacillus diphtheriæ* to guinea-pigs is not entirely satisfactory, for although definite strains of *Bacillus diphtheriæ* may prove fatal to guinea-pigs, it does not follow that these virulent strains, when harboured by a carrier, are those that enable him to transmit clinical diphtheria to other persons.

In early times, when it was recognized that certain diseases followed the routes of human travel, it was conceived that the infection of these diseases was conveyed by the traveller. This led in the first instance to the compulsory segregation of persons for prolonged periods under quarantine regulations. But the spread of infection was not stayed. Then followed the disinfection of personal clothing and belongings, at first by exposure to air and sunlight, afterwards to dry and moist heat. Experience showed that even after the most thorough disinfection of personal baggage the infection appeared beyond the quarantine and disinfecting stations. Quarantine, even when based on modern knowledge, has not prevented the introduction of plague into Australia.

## THE SEASONAL INCIDENCE OF DISEASE.

We can state without possibility of contradiction that certain diseases have a seasonal incidence. If we take two of such diseases with which we are familiar in this country, we can say that in England whooping-cough is prevalent in the first half of the year and scarlet fever in the last half of the year, but we can give no definite reason for this fact. We can say that one is a disease of the spring and the other of the autumn. Although it has been suggested that the infection of one of these two diseases, viz., scarlet fever, may be conveyed by the domestic flea, most of us believe, rightly or wrongly, that the infection is acquired mainly through the nasopharyngeal tract, and that in the majority of cases it is transmitted from person to person. But even if this is true it adds to the difficulty of explaining why one is a disease of cold weather and the other of warm weather. We know that while anthrax attacks bovine and other animals, fowls do not contract this disease, and for the explanation we are indebted to Pasteur, who showed that if the temperature of the blood of a fowl was lowered by immersing its legs in cold water the bird could not resist infection by anthrax and the *Bacillus anthracis* could be demonstrated in its blood, and that on restoring its normal temperature the *Bacillus anthracis* disappeared from the blood-stream. This experiment of Pasteur established the fact that in certain conditions the environment of micro-organisms prohibits their capacity to develop functions detrimental to animal life, but the principle concerned cannot be applied to explain the seasonal prevalence of scarlet fever, as we have no reason to believe that there is any material difference in the body temperature of man in the spring and autumn seasons. Moreover, we do not know the causal organism of either scarlet fever or whooping-cough. Scarlet fever has been shown to be a disease that is not indigenous to India, and that children born of European parents rarely suffer from this disease while in India. Though the fact may in some measure be explained by the different habits of life of Europeans in India and in temperate climates, we do not know why the disease, when once it is introduced into what in England would be a susceptible population, does not assume epidemic proportions in India. The explanation will probably be forthcoming when once the cause of the infection is known.

Before we knew the part played by the mosquito in the spread of yellow fever we were satisfied that the presence of this disease in Swansea, in 1865, was associated with the excessive heat of the summer of that year and that the infection had been brought to our shores by a vessel from the West Indies. We now know that the activity of the mosquito is dependent on the atmospheric temperature.

It is a matter of common knowledge that when small-pox occurs in England in the late autumn and continues during the last months of the year, it may assume epidemic proportions during the first quarter of the next year, the summit of the epidemic curve being reached in the second quarter of the year, after which the epidemic declines. This phenomenon cannot be attributed solely to the temperature of the atmosphere. It is on record that a destructive epidemic of small-pox occurred in Madagascar in the year 1868 at a time when the mean temperature fluctuated between 90° and 100° F., and that this disease was observed to spread in the Hudson Bay Territory in the year 1870 when the thermometer was standing at 40° below the zero of the Fahrenheit scale. In regard to small-pox we have the experience of a mild type of this disease

introduced in 1921 into the northern Midlands, possibly from America by means of imported cotton. A very moderate amount of vaccination appears to afford immunity to this disease; except in rare instances, only the unvaccinated and persons over 40 years of age who have been vaccinated in infancy and whose protection by vaccination has diminished by lapse of time, have been attacked by the disease. It has been unaccompanied by fatal results and many of those attacked have not sought medical assistance and the disease continues to manifest its presence. At the same time we have seen in 1922 a type of small-pox occur in London and its neighbourhood, introduced possibly from the East, which has exhibited a virulence and has been accompanied by a mortality probably as great as any of which this country has had previous experience.

We are still without definite knowledge of the causative micro-organism of small-pox, chicken-pox, scarlet fever, measles, typhus fever, and perhaps to this list influenza may be added. We believe that these diseases, with the exception of typhus, are transmitted directly from person to person without the intervention of any insect carrier. At one time, and until quite recently, we thought that the infection of typhus was communicated in like fashion. Now we know that the infection is transmitted by a biting insect, the louse. Poverty and want, and times of famine, have been recognized for many years as associated with epidemic prevalence of typhus and we can appreciate the fact that verminous conditions of persons are allied with such occurrences, and that the verminous condition accounts for instances that have been observed when typhus has been prevalent, and when famine and destitution have been absent. With the knowledge that lousiness must be fairly common among certain classes in Ireland, as evidenced by the fact that some 60 per cent. of Irish emigrants that arrive in Liverpool on their way to America and Canada are found to be verminous, we can realize that the absence of widespread prevalence of typhus fever in Ireland must be due to the fact that the causative agent of the disease is lacking.

If we turn to diseases which are definitely associated with a causative micro-organism we can include in the number cerebro-spinal fever, enteric fever, diphtheria, cholera, and plague, and excepting plague, each of these diseases is associated with the "carrier" problem. Whether or no there are carriers of cholera who can be classed as "chronic" carriers we have no precise information, but we know that chronic carriers occur in regard to the other three diseases.

#### THE KNOWLEDGE OF THE ANCIENTS.

Samson, in an interesting paper,<sup>1</sup> has recently reminded us of instances showing that the Ancients definitely associated rats with pestilence. That they carefully protected, and when possible, domesticated the natural enemies of the rat, even deifying some of the more useful ones such as the cat, the kestrel and the cobra. That they made special use of rat-eating species of snakes, and constructed snake-pits in the medical temples in which they kept large numbers of these harmless snakes, and were thus able to send these snakes to be liberated at places in which plague occurred; and he cites a case of the use of these snakes in Rome 293 B.C., when plague broke out in that city. That the Ancients protected the scarab because it destroyed the Heltu worm, ankylostoma, and preserved the ibis, because, its usual food being fresh

<sup>1</sup> "Tropical and Sub-tropical Diseases," *Journ. Trop. Med. and Hygiene*, 1922, xxv, p. 189.

water conivalve molluscs, it kept at bay two formidable snail-fostered parasites, *Schistosoma hæmatobium*, which gives rise to endemic hæmaturia, and *Schistosoma mansoni* which is one of the causes of dysentery. That a Babylonian clay tablet, baked over three thousand years ago, now in the British Museum, bears in cuneiform signs the name of the "Fever Fly," that a coin of Greek Sicily commemorates the elimination of malaria by drainage, that Peruvian aborigines, long before their conquest, used cinchona bark in the treatment of intermittent fevers and that the ancient Egyptians employed cotton netting as a protection against mosquitoes. Sambon mentions other instances of similar character from which he deduces that "modern nations undertaking colonization should copy the great colonists of the classic period whose success in planting large, prosperous colonies was due to the fact that their first step invariably was to banish disease by means of thorough sanitation." But his paper brings into prominence the fact that in the classic days the Ancients possessed a knowledge of the causation of disease which faded into oblivion during the Middle Ages and which is only now being brought to light.

#### THE EPIDEMICITY OF DISEASE.

To understand the individual circumstances in which diseases have occurred within the several periods of time and at the various parts of the globe, to show whether they have been subject to any differences, and of what kind according to the time and place; what causal relations exist between the factors of disease acting at particular times and in particular places, on the one hand, and the character of the diseases that have actually occurred on the other; and finally to show how those diseases are related to one another in their prevalence through time and through space, are matters that require prolonged, careful and accurate study before successful accomplishment can be achieved.

Our available knowledge does not suffice to account for recurring epidemics and pandemics of disease; of the nature of their cause, or the manner in which they operate, and of the influence that brings them about we know but little. It cannot be said that we understand the precise circumstances in which diseases have occurred at different times and in different parts of the world, and whether they have been subject to the same or to differing agencies, and if so, to what kind according to time and place.

It has been said that disease is merely the reaction of the system under the influence of causes subversive to the normal and healthy functions; and that it is clear that whenever it arises some antecedent or existing cause must have been in operation, and that the previous action of the antecedent or existing cause may be concluded from the results. In dealing with epidemic and pandemic disease, while it is evident that antecedent or existing causes must have been in operation, we cannot as yet say that we are familiar with them. Careful inquiry, while it has demonstrated the prevalence and intensity of the causes of disease at different places, has failed to reveal the influences at work which determine the distribution and behaviour of epidemics or the laws under which they prevail. Such advances as have been made in our knowledge in recent years have been due to the precision with which intensive study of different problems has been carried out, and we have seen the resulting benefit that has accrued to the human race from the application of this knowledge to the everyday circumstances of life. The comparative freedom of Central America and the Panama zone from yellow fever, and the advantages to the

trade and commerce of the world that have followed the opening of the Panama Canal, and the civilization and colonization of the Eastern world that have attended the control of malaria, are examples of this. While we know that certain diseases are prone to a cyclical recurrence, we do not yet know what causes are responsible for this recurring cycle of disease prevalence. It is not sufficient explanation to say that epidemics of measles are due to an increase in susceptible material, i.e., of children born since the last epidemic, for this would not account for the seasonal prevalence of measles, and still less would it explain the widespread and sudden prevalence of epidemics of influenza throughout the world, during which persons who have passed through a previous attack are again affected.

It may be that certain atmospheric conditions so affect the human constitution as to lessen its power of resistance to disease, or that certain low forms of life that we know to be active agents in disease production acquire an additional increase in virulence and power of propagation under differing conditions of the atmosphere not solely referable to changes in temperature. It has been observed on occasions of epidemic prevalence of small-pox that vaccine lymph has appeared to possess undue potency, as shown by the result of vaccination of persons, and that this cannot altogether be explained by a want of due care in the performance of the operation of vaccination, which is prone to be associated with panic vaccination. It may be that at such times the susceptibility of the individual to "vaccinia" or to contract small-pox is increased.

It has been urged by those who have opposed the theory of the operation of an epidemic or pandemic cause that if it existed every locality within its influence should manifest the disease or diseases it was supposed to induce to nearly an equal extent; and that this has not been found to be so. Simon wrote: "Not even the merest tiro in medicine supposes that contagion (as a morbid power acting from each sick chamber) operates equally on all persons or equally under all varying circumstances of time and place."

In medicine there has always been a strong conservative bias and an aversion to relinquish orthodox theories, notwithstanding the fact that the whole science of medicine demonstrates the necessity of studying the behaviour of disease in all its aspects, and that progress in the science of medicine is dependent on the application of knowledge acquired through observation and experiment. We have to recognize that frequently more energy is expended in opposing a new idea than in advancing it, and that this opposition may be maintained for many years after a discovery has become an established fact. The opposition to vaccination as a preventive of small-pox dies hard. This opposition to progress is not, however, limited to medicine, for hardly any great scientific theory has been accepted without strenuous opposition, the dynamical theory of heat, the theory of undulation, the theory of evolution and others, have had all to fight their way through to victory.

The belief in a living cause of disease or *contagium vivum* is very old. We cannot fail to realize that undoubtedly the greatest progress made in recent years in our knowledge of epidemiology has been the translation of this belief into an established fact; and that this has resulted from the genius of Pasteur in expounding the germ theory of the causation and course of disease, and from the discoveries of Laveran, Manson and Ronald Ross as to the part played by parasites and insect carriers in disease production. While all this serves to explain endemic forms of disease, it does not account for the rapid diffusion and widespread prevalence of disease in epidemic or pandemic form. The



essential cause of epidemic diffusion of disease must be dependent on some other factor or factors. There must be something superadded to the disease-producing organisms that augments their virulence or power to affect man; or that increases the susceptibility of the individual or diminishes his power of resistance. The nature of the influence in operation and the necessary part that it plays is unknown.

Going back to Sydenham, we find that he strongly insisted on the specific character of diseases, which he says are like plants, having each its typical way of beginning, developing, and coming to an end, and that the cause of the prevalence of disease was the epidemic constitution of the year. This epidemic constitution was something more than mere heat or cold, dryness or moisture, and it was related to certain alterations in the air.

If we go further back than Sydenham, we find that our medical ancestors had recourse to various agencies to explain the origin of disease, and to which they attributed its causation and spread. Epidemics were frequently associated with some mysterious power of the sun, moon, planets, stars, comets, earthquakes, volcanic eruptions, inundations, spasmodic convulsions of the sickened earth, a peculiar conception of the air, or an epidemic constitution of the atmosphere. One cannot fail to be impressed with the persistence of our forefathers that the condition of the atmosphere is in some way responsible for the presence or the absence of disease in epidemic form. At the same time, we must confess that meteorology has hitherto thrown little light on the subject, and notwithstanding the present delicate and far-reaching chemical and physical methods that have been employed in the investigations of the condition of the atmosphere, and the magnetic currents of earth and air, these means have entirely failed to show any relation between the physical properties of the air and epidemics of disease.

It is common knowledge that during times of thunderstorms milk readily turns sour. At such times the atmosphere may be charged with moisture, its temperature raised and so forth. But in like conditions of the atmosphere and in the absence of thunderstorms, milk remains unaffected. There appears to be no explanation of this save in the electrical condition of the atmosphere, and if this is the factor that determines the observed phenomenon, knowledge of the manner in which it acts is lacking. Can it be that the acid-producing micro-organisms are stimulated into increased activity through the electrical condition of the atmosphere?

There is another matter which has relation to this subject, namely, the preparation in starch factories of rice starch for laundry purposes. After the starch has been ground into a paste to which water is added, it is further washed with dilute caustic soda to dissolve out more nitrogenous matter (the gluten). At all stages from the rice paste to the final washing the liquid is kept alkaline with caustic soda. It is a well recognized fact that to maintain this alkalinity more caustic soda is required in hot weather than in cold, and further, that in hot weather, when there is thunder in the air, still more caustic soda is required. Under such weather conditions it is not an unusual practice to add formalin to the rice paste to prevent it "turning sour."

The late Dr. R. W. S. Bishop, writing of his experience as a country practitioner at Kirby Malzeard, in the moorland district of Yorkshire, states that "Influenza spread sometimes so rapidly, specially on the exposed tablelands, that I am perfectly certain climatic influences played a much greater part in its causation than personal contagion." Similar experiences of almost simultaneous occurrence of the disease over wide tracts of land have

been recorded by other observers, and these accounts can be considered in conjunction with narratives of ships at sea that have held no communication with the land, and on board of which outbreaks of influenza have occurred at times when the malady has been prevalent on the coasts nearest to the position of the ships. Can the explanation be that the causal agent of the disease, in harmless form, is harboured by man, until some atmospheric condition affords it virulence? Does it militate against such a theory that the disease may break out among islanders two or three days after the arrival of a ship from a foreign port under circumstances that suggest the spread of the malady by direct infection from person to person?

#### CONCLUSION.

Although from such knowledge as the medical profession has possessed at any given period, it has been argued that this or that circumstance cannot affect the prevalence or the absence of disease, yet these circumstances should be reviewed from time to time in order to ascertain whether recent discoveries in medicine or in allied subjects may supply information that in some way or another may assist to unravel one of the multitude of epidemiological problems. Manson stated his opinion that the mental horizon of the true scientific principle is determined neither by size, distance, nor social conditions. If we direct our attention to the discoveries which of recent years have followed investigation in regard to the physical and electrical conditions of the atmosphere we realize the important results that have followed the practical application of these discoveries to the business of the world. Travel for long distances through space by aeroplane is an established fact, and wireless telegraphy has revolutionized methods of communication between distant parts of the globe. Surely such discoveries should arouse a spirit of inquiry among students of epidemiology and should lead to a re-opening of the question of atmospheric influence on the prevalence, and particularly on the epidemic prevalence, of disease. The difficulties associated with such an investigation are great, and much time would necessarily be occupied in a systematic attempt to carry through such an undertaking. Though it extends to a sphere that to us may seem immeasurable and one that has only been explored to a very limited extent—for to those who made the attempt it appeared to be a barren waste—such an inquiry after truth might obtain confirmation of an anciently conceived opinion. It might result in filling in, in some measure, the scheme of epidemic diffusion of disease sketched in broad outline by Hippocrates more than two thousand years ago.

Progress in the study of epidemiology is closely associated with precise observations in the wide realm of Nature, and in the correct interpretation of these observations. This requires time and opportunity, patience and perseverance, enterprise and experience. There is nothing new or startling in this statement: it is two hundred and fifty years since Sydenham wrote: "True practice consists in the observations of Nature."

The student in epidemiology can in all humility echo the avowal made by Fracastor four hundred years ago: "I am well aware that it is difficult to say what Heaven does effect and in what fashion, and to find a certain assured cause for every event."



## Section of Epidemiology and State Medicine.

President—Dr. RICHARD J. REECE, C.B.

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### The Epidemiology of Surface Disease of the Eyes.

By N. BISHOP HARMAN, M.A., M.B.Cantab., F.R.C.S.Eng.

EPIDEMICS of superficial inflammations of the eyes and eyelids are rare in this country; when they occur they are not of a severe order, but I shall hope to supply you with evidence to prove that in almost every case their appearance constitutes a severe reflection on the habits and manner of the group affected by an epidemic. There is no sphere of medicine of which it may be more truly said that dirt and disease are synonymous than of surface diseases of the eyes. The dirty suffer, the clean for the most part are exempt. The growth of habits of cleanliness has diminished endemic "ophthalmias" and correspondingly reduced the risks of epidemics. Superficial diseases of the eyes may be regarded as a very close index of that chief feature of civilization—cleanliness, and from that point of view even so special a subject as eye disease may be of interest to your Section.

Although severe epidemics are now so rare as to be almost unknown, it was not always so. After the Napoleonic wars (1798-1810) there was in this country an epidemic of purulent ophthalmia of great severity, and a brief narration of the outstanding features of that epidemic may well form the text of this paper. A remarkably lucid and critical account by John Vetch, M.D., member of the Edinburgh Medical Society, and assistant-surgeon to the 54th Foot Regiment, was published in a little book entitled: "An Account of the Ophthalmia which has appeared in England since the Return of the British Army from Egypt," London, 1807 [1]. His statement of the case is as follows: A battalion of raw troops—52nd Regiment—who had just been raised were stationed at Hythe, and there developed a disastrous purulent affection of the eyes. "They were," he writes, "in a tolerably healthy state for a corps so lately raised. . . . Excepting a great proportion of venereal diseases, no particular distemper seemed to prevail, and the number in hospitals were rapidly decreasing when the first case of ophthalmia made its appearance." Vetch at once put out of account: "Exposure to reflection of the sun's rays from the shingle and the great quantity of fine sand which a long prevalence of blowing weather carried from it." Three other battalions located there were unaffected. He noted, however, and thought there might be in it some bearing on the case, that the diseased troops lodged in quarters used by troops similarly but slightly affected, and that these men had come from Egypt.

Then he describes the disorder with remarkable lucidity. The intensity of the onset, the severity of œdema and chemosis, the intense vascularity, the profuse purulent discharge—like urethral discharge—amounting to several ounces in the day, the "hypersarcosis of the conjunctiva, the ulceration and perforation of the cornea, the swelling of some lymphatic glands about the articulation of the lower jaw"—all these are typical of an acute gonorrhœal ophthalmia. If further evidence be needed there are the pictures published by Vetch in his two books. With one exception they are all representations of gonorrhœal ophthalmia. His description, his pictures, and this admission of the excess of venereal disease amongst the troops affected, make it impossible to arrive at any other conclusion. There are also the remarkable observations of Vetch on the liability of such subjects to a succession of gonorrhœal urethritis, purulent ophthalmia, arthritis, and iritis, an observation which Mackenzie [2] (one of the most able of ophthalmic surgeons of his day) stigmatized contemptuously as a "notion" and "a good example of hasty generalization in regard to diseases between which no other relation than that of occurrence has been pointed out."

When this severe epidemic occurred there was no knowledge of its essential contagiousness, in origin and propagation, and the general belief was that it was propagated by atmospheric conditions, miasma, effluvia, and the like. Mackenzie, in describing a horrible story of the French slave ship *Rodeur*, states that he regards it as "a fearful instance of puro-mucous conjunctivitis excited by atmospheric influence spreading by contagion." So that it is not to be wondered at that troops affected by Egyptian ophthalmia were scattered broadcast through Europe, and that the disease attacked the civil population with disastrous results. Vetch came to better conclusions and his words are worth quoting. Discussing contagion he writes:—

"No military or medical officer has yet contracted the disease without being sensible of some local communication, and the uniform escape of those of both capacities who daily inspect the sick is a sufficient answer . . . to the explanation that it propagates by some subtle effluvia arising either from the system or the eye of the patient."

"I have examined with impunity the eyes when under any particular state of the disease with a glass whose focus requires that the eye of the observer must be within less than 1 in. of the object. The opinion that the disease is communicated in the manner I have endeavoured to maintain now prevails so generally among . . . officers of those regiments in which it has occurred in England that all fear of receiving the disease in any other way is completely laid aside."

With regard to prevention he writes:—

"I have observed in many cases the disease has advanced to such a state as to form purulent matter before the patient was himself aware that he was affected. A minute and daily inspection by the medical officers becomes a duty of the first moment, both on account of the individuals who may be affected, and their comrades, by the immediate removal of the former. To prevent, however, the consequences which may accrue before the discovery of the disease, other means should not be neglected, every precaution should be instilled into the men to induce them, of their own accord, to be attentive to avoiding the disease."

"The use of barrack towels should be laid aside on the first appearance of the disease, as they afford a constant medium for its communication; at the same time, if the men are not provided with separate towels, they will, in all probability, have recourse to some other article, which they will use in common, while it will be less frequently renewed."

"Separating men as much as possible is a good precaution, though by placing them in cantonments we must effect any good at the expense of the inhabitants."

"To prevent the contagious matter from taking effect, should it have got access to the eyes, in defiance of these precautions, it will be found a very salutary practice frequently to parade the men with separate vessels of water, while an officer attends to see their faces and eyes carefully washed."

"As exposure to sun, exercise, or similar causes, cannot produce the disease unless its specific contagion has been previously applied, no danger need be apprehended from making the healthy undergo their usual fatigue, which may even have the good effect of making any cases which are latent more liable to detection by the aggravation it may occasion."

In these few abstracts which have been given from Dr. Vetch's book there is as complete an account of the essential features of an epidemic as could be desired, and the completeness of it is the more remarkable when the general pathology current at the time he wrote is borne in mind. He gives a terse yet clear description of the symptoms of the disease, arrives at a precise estimate of the nature of the contagion and its mode of transference; he notes correlated symptoms which he considers to be influential in the production of the epidemic: his judgment as to the treatment of the condition, measures for preventing its spread, and his comments on erroneous ideas, the carrying out of which he concludes would aggravate the epidemic, are as precise and sound as though they had been written to-day with a full knowledge of the microbic origin of the disease.

In these days when epidemics are so rare, even during a war of unparalleled extent when multitudes of men from every clime have been moved and removed north, south, east, and west, and brought no epidemic of eye disease in any way resembling those which affected Europe during the wars of Napoleon, it is difficult to determine the influences which may now bring about epidemics, against which we must be on our guard, by the study of such minor epidemics as may come under our experience. We must needs arrive at an appreciation of the risk by other means. This may be attained by a mass study of endemic cases, that is, of the records of cases obtainable through some sufficient period of time at some suitable place. The out-patient records of the hospitals of our larger cities furnish valuable material for this purpose. In this paper I will endeavour to give the results of such an inquiry, and show what are the influences which produce an accentuation of the liability to surface disease of the eyes, that is an accentuation of those influences which tend to make endemic disease more or less epidemic.

The cases under review have been classified so as to show the effects of:

- (1) Social conditions; (2) age liability; (3) sex liability; (4) seasonal influence; (5) the influence of other epidemics.

The material used is derived from three sources: One year's cases at a clinic in a large ophthalmic hospital, ten years' cases at a clinic in a hospital for children, both situated in London; the eye hospital takes cases mainly from its own neighbourhood, but also from all London and the home counties, the children's hospital receives cases from its own neighbourhood only. The third source of evidence has been London elementary schools.

#### SOCIAL CONDITIONS.

The effects of social conditions will be shown by reference to the cases from the eye clinic and conditions observed in the elementary schools. The patients at the eye clinic were seen in 1902, at a time when there was a

considerable migration to this country from Russia owing to the effects of war in the far East. The patients attending the clinic numbered close on 3,000 : of these 2,124 were considered to be natives and 647 aliens. The general state of the alien patients was bad, and distinctly inferior to that of the native patients. The incidence of various eye conditions amongst the two classes was as follows:—

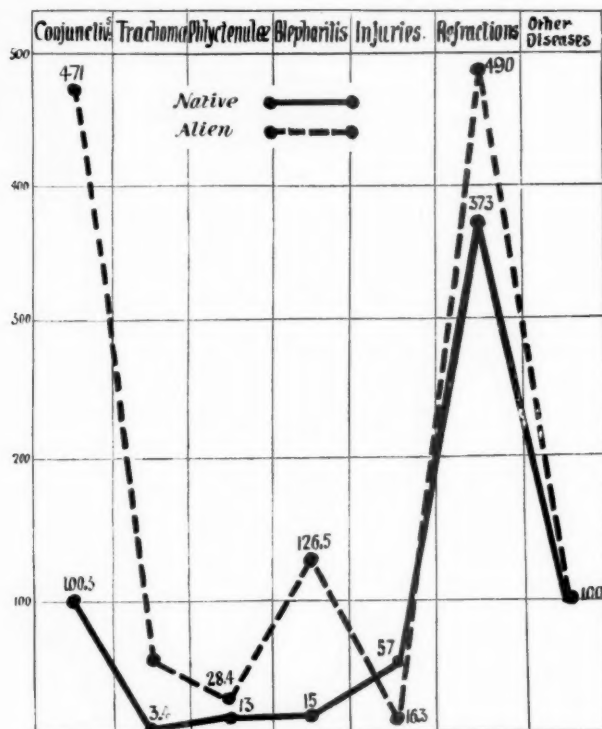


FIG. 1.—The relative incidence of eye conditions in native and alien patients. All dirt diseases are more frequent amongst the poor, ill-kept aliens.

Conditions	Cases		Ratio	
	Native	Alien	Native	Alien
Conjunctivitis ... ..	321	251	3	2
Trachoma... ..	11	22	1	2
Phlyctenulæ ... ..	42	14	3	1
Blepharitis ... ..	48	63	4	5
Injuries ... ..	180	8	22	1
Refractions ... ..	1,202	240	5	1
Other diseases ... ..	320	49	6	1
Total ... ..	2,124	647	3.5	1

The figures are striking. Surface disease of the eyes was much more common amongst the ill-kept alien patients than amongst the better-kept native patients. The native patients were not infrequently poor, dirty, and came from over-crowded quarters, yet in general there could be no doubt that they were superior in personal cleanliness to the alien patients, and the better care is reflected in their lesser liability to eye inflammations.

Between the months of July and November, 1903, I made a personal examination of all the children attending the elementary schools in the Hackney division of the London area. Thirty schools were examined, with 21,893 children of ages from 7 to 13 years. The schools were classified according to my judgment of the general condition of the region, the housing, and the evidences of care or neglect amongst the children, conditions that would be likely to affect their health. Thirteen schools were considered to attain to a fair average of cleanliness, three schools were above the average, and fourteen were below. The incidence of eye conditions in these schools, which had already been collected, was related to these general conditions of cleanliness. The following results are forthcoming:—

Cleanliness				Cases of eye disease				Percentage
Above average	...	...	...	19 in	2,174	...	...	0·873
Average	...	...	...	134 in	9,463	...	...	1·416
Below average	...	...	...	197 in	10,256	...	...	1·92
Totals	...	...	...	350 in	21,893	...	...	1·608

The clean schools were situated in the north of Hackney, where good houses and many open spaces are found; the dirty schools in Hoxton, Haggerston, and Bethnal Green, where there are miles of mean streets crowded with human beings. The schools above average cleanliness showed an incidence of disease of about one half that found for the schools below the average of cleanliness, yet it must be remembered that no one of the best of these schools was without some dirty and ill-kept children, for in every part of London there is to be found some "slum" street which breeds its quota of dirtiness. A similar investigation was made by Dr. P. J. Hay [4] in Sheffield in 1913; his figures show that children in bad districts suffer from conjunctivitis and blepharitis even to four to six times the extent that clean children are affected.

A further investigation was made in two of the London schools. One of the best in the northern area was selected, and another of the poorest in Bethnal Green. The head teachers were asked to select groups of their average scholars. These were noted to be free from eye disease. Twenty-five boys and twenty-five girls in each school were examined, five each of the ages of 5, 7, 8, 11 and 13 years. On a day succeeding several wet days, so that dust infection was eliminated, inoculations were made from the conjunctivæ of these children. Similarly the bacteriology of the lid margins was investigated in a number of healthy eyes. In one class-room of a poor school ten of the cleanest and ten of the dirtiest children were chosen who were free from signs of surface disease. The results were the following:—

## BACTERIOLOGY OF HEALTHY CONJUNCTIVÆ.

50 clean children	...	20 sterile	...	262 colonies in others
50 dirty children	...	3 sterile	...	789 colonies in others

## BACTERIOLOGY OF LID MARGINS.

10 clean children	...	None sterile	...	610 colonies
10 dirty children	...	None sterile	...	690 colonies, excess of colour cocci

The difference in the conjunctival flora is considerable, less in that of the lids; but in the latter case, all the children were in the same room and used the same books.

There is a last piece of evidence of the influence of personal care, collected before the school medical examinations and nursing inspections had wrought such a revolution in general cleanliness. The state of the follicles of the conjunctiva are almost an indicator of good health of that membrane, even as small tonsils and absence of adenoids are of the nose and throat. In 1,000 children in a fair average London school a systematic examination of the conjunctiva was made; the visible presence of these follicles was ascertained, and at the same time the visual acuity was noted, and in the girls the condition of the hair of the head. It was found that the incidence of visible lymph follicles in boys and girls was the same from the ages of 3 to 9 years; from the latter age onwards the excess in the girls was 30 per cent. This sudden and sustained increase among the girls was inexplicable until I chanced to place alongside the chart the curve of incidence of nits in the girls' heads; it was then seen that the curve of incidence of the nits was similar to that of the follicles in the conjunctiva. There is a fair average level between the ages of 4 and 9, followed by a sudden rise of 20 per cent. in the tenth year, with a slow decline in later years, almost exactly corresponding with the curve of the follicles. Inquiries amongst the teachers showed that about the age of 10 years the girls were expected by their mothers to take their share in the home duties; with this came liberty to look after their own persons, hence the increase in nits as an indication of neglect, and with this an increase of the lymphatic structure as indicative of diminished good health. It is suggested that the two facts are dependent upon a common cause, dirt, or lack of care of the body.

Dirt and disease are almost interchangeable terms. There is perhaps no range of disease which shows a closer correspondence with dirt than the surface diseases of the eyes. Prevalence of eye disease—the various forms of conjunctivitis and lid infections, may be taken as a barometer of that most distinguishing feature of civilization, personal cleanliness. And social condition would appear to exert a more potent influence upon the incidence of these diseases, whether in endemic or epidemic occurrence, than any other factor of which we have knowledge. This is true whether we consider the plague of trachoma in Egypt, where the children are fly-blown by reason of the ignorance and superstitions of their elders, or of the outbreaks of pink eye in our own public schools, which are the effect of a misplaced communism of goods, that is, toilet articles—which should always be individualistic in the highest degree. Common towels are both “common and unclean.”

## AGE INCIDENCE.

The age of patients attending the eye clinic ranged from a few hours to 80 and more years. Some method of grouping is therefore required. This has been done according to the general conditions of life. Infancy, from birth to 3 years; school age, from 3 to 14 years; young adults, from 15 to 30 years;

adults, from 31 to 55 years; elders, from 56 and over (the number of years in this group has been taken as 25 in making calculations). Where all the superficial inflammation of the eyes and lids are taken together and allotted to their proper age-groups a curve of much interest is seen. This curve has its summit in infancy, and declines steadily from that period to old age. If the figures be related to the number of the general population, it is found that the incidence per 100,000 was 22 in infancy, 20 in school age, 18 in young adults, 15 in adults and 7 in the elders. If the figures be related to the "constant" of other disease amongst the patients attending the clinic the curve shows a much steeper descent, from a figure of 1,500 in infants, to 1,100 in school age, 500 in young adults, 350 in adults, and 100 in the elders. When the figures of the children's hospital are examined the same striking decline in the incidence of surface inflammation is found. Cases are most frequent within the first year of life,

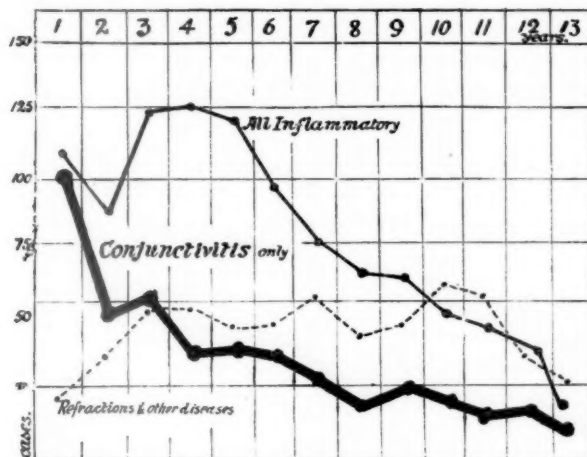


FIG. 2.—Age incidence, showing decline of liability to conjunctivitis in children. The peak of "all inflammatory" disease, years 3 to 5, is due to blepharitis or phlyctenule.

after which there is a sharp decline to the age of 13 years, the latest date at which children were seen at that hospital. In both sets of cases the decline is most marked for the severe forms of conjunctivitis due to purulent infections: with these the peak of the incidence occurs in the first few days of life, after that there are very few cases.

Exceptions to this general statement of decline from birth onwards are to be found in the curves of blepharitis, phlyctenular inflammations, and angular conjunctivitis. The separated curve of blepharitis shows a distinctive feature. There were no cases in the first nine months of life; a few rare cases appear at the close of the first year, there are a few cases also in the second year, the numbers are more than doubled in the third year, and again doubled in the fourth year. That year shows the peak of incidence, afterwards there is a rapid decline until at the seventh year the incidence is as low as in the second year. Subsequently there is a slight rise in the eighth and ninth years, followed



by a slow decline. Again it is possible to separate the curve of incidence of phlyctenular eruptions from the general curve of surface inflammations. No cases were found in the first year of life, a few appear in the second, and after that there is a sharp uprise to the fifth year, with a subsequent steady decline until cases are rare in the thirteenth year.

These features of the curves of surface inflammations distinguish blepharitis and phlyctenular eruptions from ordinary conjunctivitis. Conjunctivitis is simply a dirt disease; infants are more liable to dirt infections than elder children and adults, just because they are helpless in their early months, and then become crawlers, whose attentions are attracted by any object which can be conveyed to mouth and face, unless the care of the mother be more than ordinary. The difference in the incidence of blepharitis and phlyctenular inflammation must be sought in other directions. There is no more potent cause of the onset of blepharitis than measles. It is the habit of mothers to date the onset of some chronic trouble to some particular incident; the connexion may or may not be one of cause and effect. But in the case of blepharitis, careful observation has led me to accept the causative influence of measles as pre-eminent. With an attack of measles there is nearly always some conjunctivitis and a definite affection of the lid margins, the hair follicles and the glands that supply the needs of the lids. If there be not scrupulous attention to cleanliness during recovery from the fever, the acute attack of the lid membranes is likely to remain as a chronic blepharitis. That brings us back to the common denominator of dirt. The child with measles who is kept clean rapidly gets over the blepharitis, the dirty child does not. There is certainly another and aggravating factor, the influence of error of refractions. Eyestrain is undoubtedly a predisposing factor in the continuance of blepharitis, it may even be the cause of its onset in some cases, but it more often aggravates an existing inflammation. Here again dirt is the controlling factor, the clean will escape the risks of blepharitis, even after an affection through measles or other fever, and even though there be unrelieved eyestrain; whereas the dirty will suffer so badly that a state of the lid margins is produced that is unsightly in the highest degree and ultimately irremediable.

With the phlyctenular keratitis there is sometimes a somewhat similar association of origin in one of the exanthemata. But in noting the site of origin of these phlyctenular eruptions in a long series of cases I found that there was a "site of election" for the eruptions. The great majority of the lesions—over 75 per cent. of the original lesions—occurred at the limbus of the cornea in the lower and outer quadrant. This rim of limbus is richly supplied by nerve fibres derived from a loop formed between a branch of the lacrymal branch of the first division of the fifth nerve and the orbital branch of the second division of the fifth, the main source being from the latter branch. This innervation links up the most sensitive rim of conjunctiva with the dentition of the upper jaw. The peak of incidence of the phlyctenular eruption rises between the ages of 3 to 8 years with its summit at the fifth. The condition of the mouth of poor ill-kept children during these years surely provides irritation enough to account for the ocular trouble. Again, the same terminal branch which supplies the site of election for the phlyctenular eruptions also supplies the upper lip and side of the nose, and the frequency with which sordes of the lips, the result of chronic catarrh, are associated with phlyctenulae is notorious.

Note must be made of the fact that some observers have attempted to

associate phlyctenulæ definitely with tubercle, either as a direct or indirect effect of that infection. Possibly there are cases which show this association; a child affected with tubercle is more than ordinarily likely to suffer from phlyctenulæ. But when the life history of the bulk of these eruptions is studied a real causative association with tubercle is rendered in the highest degree unlikely, at any rate for the vast majority of the cases. At the children's hospital it was found that of all the cases one-half were cured in one week, one-fourth in two weeks, one-eighth in three weeks, one-sixteenth in four weeks, and one thirty-second linger on for six weeks. The remaining fraction of one thirty-second of the total cases forms the chronic group, with a severe relapsing keratitis which proves very refractory to treatment in the usual home conditions of the child, but which rapidly responds to medicinal treatment if only the child be removed to healthier surroundings with cleanliness and good food.

There is one point in the liability to phlyctenular affections which is of interest. Jewish patients, even among the poorest and least well kept of the east-end population, were only in rarest exceptions sufferers from the disease. They showed an excessive liability to dirt diseases, but were almost exempt from phlyctenular disease. It is likely that the clue to the lesser liability of the aliens is to be found in food. The staple food of the native poor is tea with bread and butter; the staple food of the alien is richly impregnated with oils and to an extent we could not tolerate. The best medicinal treatment—other than environment—for phlyctenulæ is cod-liver oil, and possibly what is the best cure is also the best preventative. In this one instance dirt is not omnipotent, it plays its part without doubt, the dirty, septic mouth is the primary source of irritation which brings about these herpetiform eruptions, but even that influence may be overridden when the food is so good as to secure a high state of resistance in the body generally.

There is one other exception to the general rule that conjunctival infections are most common in early years, and that is in the case of angular conjunctivitis, due to the Morax-Axenfeld bacillus. Cases are scarcely known amongst infants, there are few in the school age, but there is a steady increase of liability through the young adult and adult groups with a maximum amongst the elders. The figures of the eye clinic are: Infants 0, school age 5, young adults 19, adults 24, elders 30; that is the number of cases in 3,000 patients seen in one year. The organism is almost parasitic, the disease is chronic, and never found in well-kept persons.

The conclusion of the matter of age liability to surface disease of the eyes is the same as that found for social conditions—dirt is the chief factor.

#### SEX INFLUENCE.

The curves of incidence of surface inflammations of the eyes for males and females run an almost parallel course for the age groups of infancy, school years, and elders, the female patients being rather in excess of the males. A difference is found in the young adult group; there the incidence is a good deal higher amongst the males than among the females. The explanation is probably to be found in the character of the work done by the two sexes at the most active period of life. If the curve of injuries to the eyes from foreign bodies and the like, be examined, it is found that the curve of injuries for females forms an almost uniform level for all age-groups; that for the males

conforms to this for infants, school age, and elders, but for the young adults there is a sharp uprise, so that whereas in this age-group the females show a relative incidence of 25, the males run as high as 350. It would seem that there must be a connexion between these two curves. A liability to injuries is almost certain to be associated with an almost equal if not greater liability to attacks of conjunctivitis due to slighter injuries which infect the conjunctiva without calling for the active removal of a macroscopic foreign body. The parallelism of this uprise of the male incidence in this age-group of young adults for both conjunctivitis and injuries would appear to be more than mere coincidence.

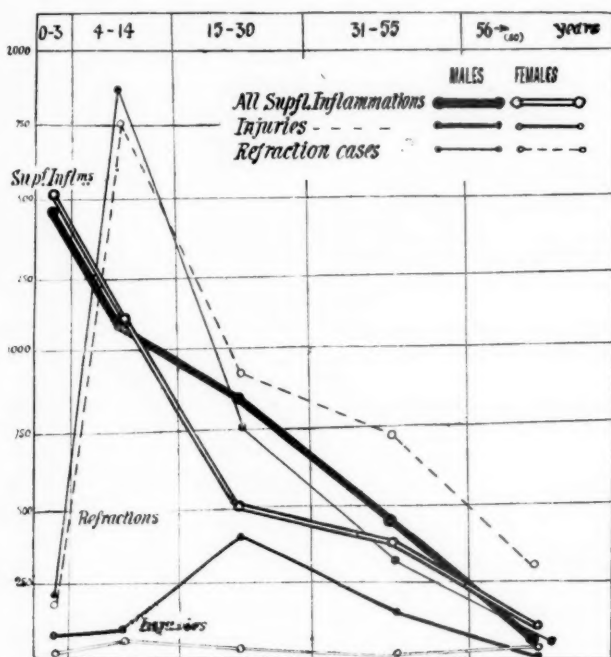


FIG. 3.—Sex influence. Females more generally affected, except for injuries

Besides the evidence of these curves of the greater general liability of females to conjunctivitis there are certain figures relating to specific forms of conjunctivitis. Angular conjunctivitis, due to infection with the Morax-Axenfeld bacillus, is more frequent among women than men, it occurred in 14'91 per 1,000 of women patients and 12'15 of men. Muco-purulent conjunctivitis, due to infection with the Koch-Weeks bacillus, occurred in 11'6 per 1,000 of women patients, and 6'12 of men. To what influence these specific differences may be assigned we can only speculate. There is no evidence that the women patients of the hospital class are less well kept than the men.

They are probably more indoors than the men, which may render them less resistant to infection. There is, however, one difference in habits which may be held accountable, the greater use of seated sanitary conveniences by the women. Is it possible that the higher incidence of surface infection amongst females is a reflection upon the upkeep of these sanitary conveniences amongst the poor?

#### SEASONAL INFLUENCE.

The influence of atmospheric conditions has a more than academic interest. At one time ophthalmic surgeons were much exercised about the ætiology of "membranous" conjunctivitis. Was every case of this sort diphtheritic, or were other and less constitutional infective organisms equally responsible? One strong argument of the supporter of a general diphtheritic

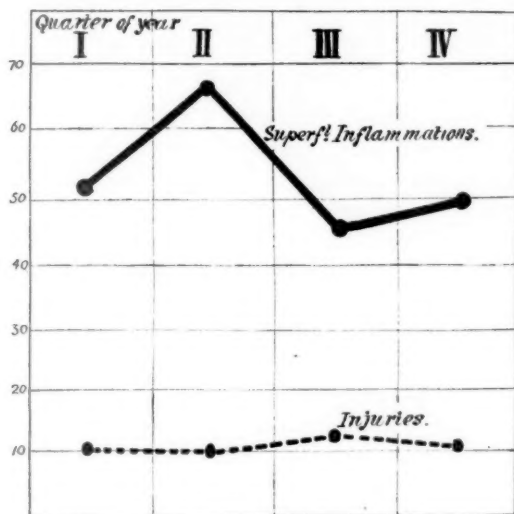


FIG. 4.—Seasonal influence. Surface inflammations are at their maximum in the spring quarter.

affection was the fact that the cases of this observer occurred during the months of April, May and June, the period, he said, in which faucial diphtheria was most prevalent. When I came to work out the seasonal incidence of the conjunctivitis it was found that all forms were more prevalent in these same early summer months than at any other period of the year. The same finding was obtained from the cases of the eye clinic and of the children's clinic. The eye clinic shows an almost equal incidence for the first, third and fourth quarters of the year, running between 90 and 105; for the second quarter there is an uprise to 150. This is in sharp contrast with the curve of incidence of refractions and other disease, for with these the peak occurs in the dark winter months of the first quarter, with a sharp fall

for the second quarter. An identical seasonal curve was found for the children's cases: first, third and fourth quarters very nearly the same, running between forty-eight and fifty-two cases, whilst the second quarter showed an uprise to sixty-eight cases. The curve of injuries, on the contrary, showed an almost uniform line of ten cases each quarter. The reason for the excess incidence during the second quarter of the year must be due to the onset of the windy weather. The streets are drying rapidly, dirt is blown into the eyes and infections ensue; each of the minute but numberless foreign bodies that are blown into the eyes carries a host of organisms with it and may set up an inflammation. These conditions were possibly more potent for evil at the time when these figures were collected, motor cars were then scarce, and horse traffic more abundant.

There was found to be one exception to this seasonal variation in the incidence of superficial eye inflammations: phlyctenular conjunctivitis and keratitis show little variation through the year, and no peak such as shown by the inflammations due to direct infections of the conjunctiva.

Mr. A. F. MacCallan [5], director of ophthalmic hospitals in Egypt, has devoted much attention to seasonal influence. In his latest and ninth report of 1921, he gives some interesting curves showing the relation of temperature to the number of patients treated, also he gives the figures of the number of cases in which the infective organisms of various forms of conjunctivitis are found in the monthly returns. The study of these charts and figures can leave no doubt that there is a definite causal connexion between temperatures and liability to conjunctival infections. In Egypt the peak of incidence is in July, which is nearly at the height of the summer temperature; the difference between the number of cases seen in the winter and summer months is enormous, in January the cases fall to 6,000 and rise in July to 14,300. Similarly the positive bacteriological findings numbered in January 337, and in July 2,135. The findings of seasonal influence again point to the dominance of dirt as the factor in the production of conjunctival infections, dust in London and temperature in Egypt, where warmth favours dirt.

#### THE INFLUENCE OF OTHER EPIDEMICS.

The influence of measles in the production of blepharitis and conjunctival infections has been noted. Other of the febrile diseases may be equally fertile of infection, and in this connexion it may be of interest to note the extent to which the exanthemata are responsible for the occurrence of purulent conjunctivitis and loss of sight. Purulent conjunctivitis is most common directly after birth. One child in every hundred born becomes infected with organisms of so virulent a character and suffers a real purulent inflammation, and most of these cases are due to the gonococcus. After this initial outburst at birth purulent inflammations are few, and occur almost solely amongst children who are at the time suffering, or who have just suffered, from one of the exanthemata. In figures collected from the London Blind School there were 367 cases of blindness due to ophthalmia neonatorum and 90 cases due to purulent conjunctivitis of later years. Of these, 30 were due to causes unknown, that is, the date of origin was known but the actual cause was not ascertainable; 18 were due to measles, 9 to streptococcal infections, 8 to scarlet fever, 6 to meningitis, 4 each to small-pox and trachoma, 3 to diphtheria, 2 each to pneumonia, gonorrhoea and whooping-cough, 1 each to German measles and chicken-pox. There is no lack of recognition of the disastrous

effects of ophthalmia neonatorum, but there is less knowledge that one-fourth as many cases of blindness occur in the early years of life from such common causes as measles, scarlet fever and the like. These cases are almost always as preventable as is ophthalmia neonatorum; scrupulous cleanliness and care of the eyes when any sign of conjunctivitis appears will in most cases prevent such untoward a happening as a severe purulent affection. Certainly no such cases as blindness after meningitis should be seen, for those are due to sheer inattention to the eyes when the patient is in a state of unconsciousness.

I close this paper by giving two quotations from the Report of the

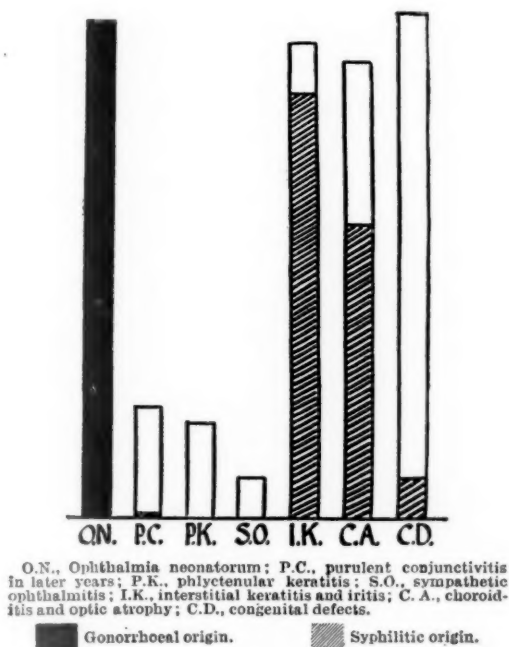


FIG. 5.—The causes of blindness in school children, under 16 years.

Departmental Committee on the Causes and Prevention of Blindness [6] issued last year. The paragraphs show from the evidence of past experience how successful have been efforts to prevent the worst consequences of conjunctival disease and the lines upon which still further successes may be assured. On the incidence of ophthalmia neonatorum and its effects the Committee report:—

“ We have clear indication from the figures submitted to us by Mr. Bishop Harman with regard to school children in London that the proportionate incidence of blindness due to this disease has markedly decreased. These figures show that between 1907

and 1913 (inclusive) there were admitted to schools for the blind 737 children, of whom 134 were blind from ophthalmia neonatorum, or 18.03 per cent. The number admitted between 1914 and 1920 (inclusive) was 755, the number blinded through this disease 89, a percentage of 11.91, a very material diminution. Again, if the figure for the first period of 134 children blind from ophthalmia neonatorum be related to the average of the elementary school roll in London over the years 1907-13, viz., 731,981, this shows blindness from this cause in 18.3 per 100,000 children, and if the figure 89 of the second series be related to the average roll over the years 1914-20, viz., 717,665, the incidence is 12.5 per 100,000 children."

At the conclusion of the report there are these paragraphs:—

"Certain factors, predominant in the production of blindness in former days, as small-pox and trachoma, both dependent upon infection, have now been checked, and we have little doubt that a later generation will similarly view some present causes. Ophthalmia neonatorum and syphilis, predominant to-day, are also dependent upon infection, and although the problem of dealing with them is more complicated, success in the campaign against venereal disease is, in our view, the key to the prevention of blindness from these two causes."

"The perfecting of medical diagnosis and technique will effect much, as will the success attending public health activity directed against unhygienic environment and conditions inimical to health. Such factors as are associated with conditions of social life, unhealthy habits, malnutrition, &c., cannot be rectified immediately but only by the slow and gradual process of education and the attainment of a higher standard of life among the people."

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#### DISCUSSION.

Sir WILLIAM HAMER said he was greatly interested in hearing about the suppression of the barrack towel by John Vetch. The greater liability of young girls to certain eye diseases was in keeping with the corresponding greater incidence in diphtheria, which had been attributed to care of the younger children and household duties. There was, moreover, the observed greater incidence upon young girls in phthisis, of which several explanations, none of them entirely satisfactory, had been given.

Dr. R. DUDFIELD said that his experience of ophthalmia was limited to the disease in new-born infants. Judging from some preliminary inquiries he made two years ago but had been unable to complete, he thought that ophthalmia neonatorum if not actually increasing in this country was certainly not showing any notable diminution in prevalence. Fortunately, however, no records of damage to the eyes of the sufferers had come to his knowledge in his district. Mr. Harman's charts indicated that the disease increased in prevalence with increase of air temperature. Was there any suspicion of the disease being spread by flies? The manner in which flies infested diseased eyes in the East was well known. It had also been suggested that the rise in prevalence which began in the spring was in part due to road dust, &c., lifted by the high winds usual at that time of the year. Within the last seven or eight years tar-bound roads had very largely replaced water-bound roads. In his district that change was



complete. It would be interesting to know if there were any indication of a lessened prevalence of eye disease which could reasonably be attributed to the diminution in the amount of road dust consequent on the change in mode of road-binding. The extensive use of motor for horse-drawn vehicles was another factor which should be considered.

Dr. MAJOR GREENWOOD said that Mr. Harman's paper was a valuable and interesting contribution to practical epidemiology. In these philosophical days it was refreshing to have a clear and cogent statement of the evidence that dirt *was* an ætiological factor of epidemicity, of the dissemination of horrible forms of illness. With reference to the seasonal factor, he suggested that wind movement, rather than temperature, might be the important meteorological element. He desired to dwell upon one point in Mr. Harman's paper, viz., the long latent period between the discovery of truth and its dissemination. To those who, like himself, were not familiar with the special history of the facts, it was remarkable how slowly Vetch's teaching became a rule of practice. He had recently had occasion to read a work on venereal diseases, published in 1829, by John Bacot, great uncle of Mr. Arthur William Bacot, whose tragic loss members of the Section had so recently sustained. John Bacot was familiar with Vetch's work and he gave a rather amusing example of gonorrhœal ophthalmia which was worth quoting. The subject was a medical student of Trinity College, Dublin. His teacher, a distinguished surgeon, noticed that the youth had a green shade over his eyes in lecture and inquired the reason. "The tale was soon told. Mr. — had unfortunately found himself affected with gonorrhœa, had incautiously transferred the matter to the eye, and the inflammation then visible was the consequence." The lecturer, "feeling a kindly interest in the situation of his pupil," examined the eye and urged the lad "to retire immediately to his chambers and submit to the abstraction of at least thirty ounces of blood from the temporal artery, and to adopt other decisive measures for subduing the disease, which he thought proper to indicate." The student retreated in disorder from the lecture room and on his way home encountered an eminent physician who "strongly assented to the expediency of the proposed remedies and insisted on the necessity of their instant adoption, as the only means of averting the so imminent danger." Fortunately for him, however, Mr. — also met a Dr. Ridgway, who, to quote his words: "hesitated not to declare my conviction of their absolute inutility (i.e., of the blood-letting and other decisive measures) and reminded the student of the nitrate of silver, which he had often heard me expatiate on, and which I affirmed was, in my belief, the only one to be relied on with safety." The student adopted Ridgway's treatment and the end of the story was as follows: "The next day the student appeared without the green shade, as usual, before the lecturer, who expressed his surprise and pleasure at the happy change, attributing it to the prompt and effective employment of the decisive remedies he had recommended the day before. The student, however, without hesitation, declared that he had not resorted to the use of one of them; and, on being further interrogated as to the means by which the disease had been so speedily removed, avowed that this had been effected by the introduction into the eye of the solution of nitrate of silver 'by my hands the preceding evening.' Upon hearing this account, the astonishment of the lecturer showed itself in the emphatic observation of 'Well, I wonder what we shall come to next.'" He (Dr. Greenwood) was almost as much astonished as the lecturer to find within a few pages of this instructive story, the following remarks by John Bacot himself: "Recent observers have, however, been induced to believe that independently of this cause (suppression of the discharge), it (ophthalmia) may be produced by actual contact of gonorrhœal matter to the eye; and Jesse Foot engages in a long, and, I think, very needless discussion, to show, that although the inoculation or introduction of this matter can, and does occasionally, give rise to the disease, that the patient cannot infect himself from his own gonorrhœa; for, he says, if this were the case, scarcely any man or woman having that disease could possibly escape the ophthalmia here described. He, therefore, infers that it never takes place unless the matter introduced be that derived from another subject. I am much inclined to believe in this explanation, which is viewed in the same light by

Dr. Vetch." The only effect produced upon the mind of John Bacot by the story of the Trinity College student's treatment was to lead him to say that "I should almost feel inclined to make trial of it, considering the numerous instances of failure which I have witnessed or heard related by pursuing the practice hitherto recognised."<sup>1</sup>

Mr. BISHOP HARMAN (in reply) said that during the recent war there had been striking evidence of the good effects of measures of preventive medicine in the suppression of trachoma amongst the Chinese Labour Corps; the work done in this respect by Lister and Cunningham was an example of how things should be done. The effect of common towels might be best seen nowadays in elementary schools. In one case where an epidemic of muco-purulent conjunctivitis broke out in a large day school managed by a "sisterhood," the withdrawal of the towels checked the spread of the epidemic except in one class. Inquiry showed that this class was doing a piece of fine needlework, and to safeguard this the sister-in-charge gave the girls "a piece of clean linen" with which to wash their hands before doing the work. That piece of common washing material was responsible for the continuance of the epidemic.

He agreed that more cases of ophthalmia neonatorum were being notified; this was to some extent due to the greater care exercised. Returns differed widely, and this arose from differences of methods of notifications. From some districts there came exceptionally high returns; inquiry showed that these figures were derived from uncorrected information given by midwives. No case should be considered to be officially notified unless it had been seen and verified by a doctor.

The seasonal influence of conjunctivitis in this country was without doubt due to wind-borne infections; he did not think that flies had anything to do with it; and in this country warmth was of little account. Just now when the bitterly cold east winds were blowing there was more than ordinary prevalence of conjunctivitis.

"A Treatise on Syphilis," by John Bacot, Lond., 1829, p. 137.

## Section of Epidemiology and State Medicine.

President—Dr. R. J. REECE, C.B.

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### The Ultravisible Viruses considered from an Epidemiological Point of View.

By Sir WILLIAM HAMER, M.D.

THE ingenious author of "Flatland" has described the gradual opening up of the vista as we ascend from the realm of Pointland, the abyss of No Dimensions, where a little soliloquizing creature fills all space, and is the One and yet the All in All; and then emerge into Lineland, where no one ever sees anything but a point, and no Linelander can ever pass another; as we further achieve breadth also, and enter Flatland with its uneducated women, devoid of memory and of foresight, and its doctrinaire Circles (or Priests); and so at length, by an impulse "Upward not Northward," proceeding from within us, as from the arbitrary visitation of a Magician who could play tricks with one's very stomach, we come to Spaceland. Yet here we cannot rest, for as in one dimension the moving Point produced a Line with *two* terminal points: and in two dimensions the moving Line produced a Square with *four* terminal points; and in three dimensions a moving Square produced that blessed being a Cube with *eight* terminal points; so in four dimensions shall not a moving Cube—alas for analogy and also for the progress of Truth if it be not so—shall not the motion of a divine Cube result in a still more divine organization with sixteen terminal points?

We seem to have here a picture of the evolution of the germ theory during the last sixty or seventy years. From Pasteur's discovery, that fermentation was a phenomenon of life, down to the heyday of the eighties, the causal organism was the One and yet the All in All. Then the fact was recognized that the host as well as the parasite had a part to play. Next Pettenkofer and Hueppe and their schools claimed that the pioneer work of the localists demanded notice. It is now beginning to be appreciated that even three dimensions are not roomy enough. "A. Square," the author of "Flatland," when he hit upon the formula "Upward not Northward," dreamt he could supply the clue to the enigma. It occurred to him that the child of one of his pentagonal sons, a young and docile Hexagon with a mathematical turn, would be a most suitable confidant; but, even as he attempted to enlighten his precocious grandson concerning the idea revealed to him in a dream, the herald's "Oh yes! oh yes!" was heard in the street outside, proclaiming the Resolution of the Council condemning all those who tried to pervert the minds of the people by delusions, or who professed to have received revelations from

another world. This scene was painfully present to my mind last Christmas when I was approached on the subject of the present paper by those estimable Hexagons, the then secretaries of this Section.

In trying to meet their requirements it was impossible for me to follow "A. Square's" example and plunge straightway into a world of four dimensions, but I have taken advice and tried to make use of the "eye that discerns the interiors of things." Hitherto, oddly enough, despite all that has been done with the atom, the anatomy, physiology and parasitology of ultramicroscopic germs have been shamefully neglected. This line of inquiry has been rendered particularly alluring since the time when it was borne in upon the Section that Sydenham never absolutely ruled out "mutating ultravisible viruses." These last named organisms may be said to date from Buchner's demonstration in 1897, of an intracellular enzyme in the yeast cell; for it gradually became appreciated from then onwards that the rôle of the enzyme and not that of the yeast cell is comparable with that of Hamlet in the play. Then followed de Schweinitz's dethronement of the hog cholera bacillus and the definite establishment of the sovereignty of the ultravisible virus. There speedily emerged, thereupon, the two following presumptions:—

(1) That beyond the limits of visibility under the microscope (now placed at  $10^{-5}$  cm.) there exist extremely minute living forms—thus below the *microns* ( $10^{-3}$  to  $10^{-5}$  cm.) of Zsigmondy's nomenclature, there are *submicrons* (down to a limit of  $5 \times 10^{-7}$  cm.) and yet lower still *amicrons*, reaching ultimately to within the range of dimensions of molecules ( $10^{-8}$  cm.). It is claimed, in fact, that just as the advance made in the nineteenth century in physics was in the main due to the application to molecules and atoms of laws demonstrated upon bodies of a size which permitted them to be weighed and measured, so, since the laws of life hold sway within the limits of the ultravisible, they must be investigated there.

(2) That the germs of the bacteriologist assume importance in disease mainly by reason of their being able under certain conditions to live in symbiosis with the generally speaking much more deadly though far more minute ultravisible living particles. There are, in fact, it transpires, important inter-relationships between the ultravisible germs and the bacteria. These have been compared to those between man and the tools he works with; or alternatively to the partnership, on a far vaster though probably really less important scale, between alga and fungus connoted by the term lichen; perhaps the closest parallel instance of all may be found in Keeble's "Plant Animals," the simple worms *Convoluta roscoffensis* and *Convoluta paradoxa*, study of which has thrown such novel light upon parasitism generally, and in particular has suggested momentous possibilities in the world of ultravisible particles.

The facts may perhaps, here, profitably be recalled that Keeble's worms inhabit the seashore of Normandy and Brittany between tide marks; the first named species contains a large number of green cells (*Zoochlorella*), the second yellow-brown cells (*Zooxanthella*)—parasitic cells—"which have abandoned their free and independent modes of life, and have taken up their abode in the tissues of animals." Haberlandt has suggested that the cells are not, however, complete cells, "but merely chloroplasts, and that like chloroplasts of the green plant they are transmitted as colourless particles (leucoplasts) from the organism to its eggs." "It is easy to imagine, with Haberlandt," says Keeble, "that in some remote past algal cells came to exist in symbiosis with colourless *Convoluta roscoffensis*; that the animal offered such a congenial

lodging as to induce the algæ to give up going out altogether . . . .” “the original green cells become reduced to mere chloroplasts. . . .” He here finds a hint even of the origin of higher forms—of lichens, even of the green tree with trunk and branches. Into the truth of “this alluring picture, evoked by siren-voiced hypothesis,” he proceeds to inquire further.

We must, however, press upon our way, and two lines of inquiry forthwith present themselves. First, are the germs hitherto regarded as causal really primarily concerned with fermentations and with diseases, or merely concomitants, i.e., associated organisms? and, second, the question arises as to the status *qua* organization of the ultramicroscopic “enzymes.”

The former inquiry may be illustrated from the writings of Lister, who was investigating the activities associated with the growth of *Bacterium lactis* in souring milk. In his address “On the Nature of Fermentation” (*Quarterly Journal of Microscopical Science*, April, 1878),<sup>1</sup> he explains how he satisfied himself that the bacteria themselves were the cause of the fermentation. By employing the method of dilution (with boiled water) he inoculated boiled milk with drops tainted with soured milk, drops estimated to contain only one, two, four, &c., bacteria. In the case of ten drops “calculated to have on the average one bacterium each” . . . “exactly five, it so happens, have remained fluid without any curdling.” He argues “the ferment, of whatever nature, is not in solution but in the form of suspended insoluble particles. . . . The fact that some drops were destitute of the ferment proves that the ferment was not in a state of solution.” Then he asks, was the *Bacterium lactis* the cause of the fermentation or a mere concomitant? He says: “Now suppose we admit, for the sake of argument, that the lactic acid ferment consisted of particles of some non-living substance capable of multiplication as rapidly as the bacterium, but not living; a strange hypothesis, no doubt, but suppose we assume it.” Having put up this “inconceivable” thesis of lifeless multiplying particles, Lister promptly demolishes it. It “is inconceivable that, if mutually independent,” the lifeless particles and the *Bacterium lactis* should accompany one another in pairs, that “invariably where there was a *Bacterium lactis* there would be a ferment particle, and where there was no *Bacterium lactis* no ferment particle. That would be a thing as inconceivable as the other.” . . . His conclusion is: “Therefore, we have two inconceivables, one of which would have been sufficient to show that we cannot admit any other hypothesis than that the *Bacterium lactis* itself is the cause of the lactic acid fermentation.” We clearly discern, however, today that these two inconceivables might, by invoking the aid of mutation, be transformed into two unavoidable assumptions; if only it be postulated in fact that there is symbiosis between the *Bacterium lactis* and a living enzyme.

In his address “On the Lactic Fermentation and its bearings on Pathology” (*Transactions of the Pathological Society of London*, 1878),<sup>2</sup> Lister restated his thesis with even greater emphasis. Let us assume, he says, “that it is possible for insoluble particles to exist devoid of vitality, yet capable of multiplying like the bacteria. Such a notion is unsupported, I believe, by a tittle of scientific evidence; but for the sake of argument let us for a moment assume it. We should then be obliged to suppose . . . that these hypothetical particles, though merely accidental accompaniments of the bacteria, were present in precisely the

<sup>1</sup> “Collected Papers of Joseph, Baron Lister,” i, pp. 335-352.

<sup>2</sup> “Collected Papers of Joseph, Baron Lister,” i, pp. 353-386.

same numbers, a thing which is utterly inconceivable. But we should have to go further and suppose, what is equally inconceivable, that these bodies of different natures, though mere accidental concomitants, were not only exactly equally numerous, but invariably accompanied each other in pairs; so that when a bacterium was introduced into one of the glasses it was always associated with a particle of the hypothetical true ferment, and whenever the bacterium was excluded, the hypothetical ferment likewise failed to enter." . . . Hence, he concludes, *Bacterium lactis* "is the cause of the special fermentation." The odds against the erroneous doctrine are infinity  $\times$  infinity. And yet—the little more and how much it is—only assume the "non-living" particles to be living, ultramicroscopic particles, and convert the accidental concomitancy into necessary symbiotic association, and thanks to de Schweinitz and to Buchner, and the latter day Massinis, Reiner-Müllers, Tworts, and Penfolds, we have a working hypothesis.

Lister's comments on the facts that *Bacterium lactis* is not the only bacterium that sours milk, and that *Mucor racemosus* as well as yeast produces alcoholic fermentation, are very interesting. Also his distinctions between "true and pseudo-fermentations." The essential property of the true ferment, he declares, is that it is capable of self-multiplication. He alludes to speculations which might reduce the bacteria to the position of "mere accidental concomitants." Furthermore, he stresses the differing phenomena of growth of yeast in shallow and deep vessels, and hints at a distinction between fermentative and vegetative activities. He even formulates (p. 379) a theory of alcoholic fermentation which "would be a combination of the views of Pasteur and of Liebig; and while assigning with the former authority, the primary and essential place to the growing organism, would admit with the latter a catalytic influence, exerted by decomposing organic substances upon unstable compounds in their vicinity." Finally (pp. 382, 383), he argues, "it is far from being impossible that there may exist ultramicroscopic organisms, as real, as distinct, in structure, and as potent in their effects, as is the *Bacterium lactis*." This bold anticipation of the ultravisible viruses is one of the most notable of many remarkable features in these stimulating papers of Lord Lister. Lister's original thesis was that fermenting capacity was so bound up with the organism, bacterium or bacillus, as to be inseparable from it, but he appreciated that some of the seemingly anomalous results might be explained by distinguishing between vegetative and fermentative powers. He saw as in a glass darkly the phenomena now familiarly described as those of "sporting" or "mutation" in bacteria, and clearly discerned that ultravisible particles might play a part in these. He almost anticipated the discoveries of zymase and of the filter-passing hog cholera virus, yes, and even the later work of Twort and d'Herelle and Bordet. He firmly held, moreover, the view that the ultravisible particles were living organisms.

This brings us to our second line of inquiry. Can any light be thrown upon the nature of life at this abysmal level? It may be safely assumed to be unjustifiable to pass death sentence upon all particles less than  $10^{-5}$  cm. (linear dimensions); there is in truth range of sufficient amplitude, from there down to molecular dimensions ( $10^{-8}$  cm.), to give elbow-room for a great variety of living types; and present-day workers may reflect with reverence and wonder that they are "the first that ever burst into that silent sea." In thus voyaging into the unknown it may be well to reflect that the living forms encountered may have very different properties (as regards, for example, resistance to heat and chemical reagents) from those of the germs we think we know. Dante, in the heaven of the moon, when he gazed upon newly revealed personalities,



mistook them for reflected images, and looked behind him and away from the originals to see, as he supposed, the real beings themselves. This allegory may perhaps profitably be applied in the case of enzymes.

Dr. H. M. Vernon has remarked that few subjects have attracted so much attention within recent years as that of enzymes, particularly intracellular or endo-enzymes. He adds that Duclaux suggested that "the life of bacteria was nothing more than the sum total of the activities of the enzymes contained within them." For the epidemiologist interest centres upon the so-called "enzyme" which can live as it were in symbiosis with pathogenic organisms, which can multiply, *pari passu*, with its temporary host, and leave the latter so that it ceases to be pathogenic until reactivated, possibly by animal passage. How does such a conception square with the theories of biochemistry? Dr. Vernon is disposed to place "enzymes and other protein like bodies, formed and secreted by living substance," in an intermediate position between "the ordinary protein molecule" and the living organism. They "must evidently possess certain groupings which are not present in the inert protein molecule and which confer upon them their lability and their catalytic powers."

When the ultraviolet particle, unaccompanied by a schizomycete, first dawned upon the inward eye, and was satisfactorily differentiated from the "dead," "chemical," "soluble" enzyme, it was presumed to work by means of "an indwelling force whose nature is still unknown." Berzelius distinguished between catalysis and ordinary chemical action. Study of "catalysis" soon leads the student of fermentation in its relation to disease to appreciate fully a remark by Dr. Stewart in his "Recent Advances in Organic Chemistry." He says: "When we examine the matter closely we find that the foundations of theoretical organic chemistry are a series of labels by means of which we endeavour to conceal our ignorance of the fundamental phenomena of the subject." The epidemiologist, like his master Sydenham, has always been a bit of a rebel, but the more docile pathologist has, as a rule, taken "catalysis" lying down. Even the laboratory workers were startled, however, by Buchner and de Schweinitz, into realizing that, like King Canute's courtiers, they were assuming a wholly indefensible attitude when they declared that life could only reach down to  $10^{-5}$  cm. (linear dimensions) and no further.

The ground having been thus cleared, investigation may be undertaken of the life history of the simplest known fission fungi considered not as completely fashioned and immutable forms, but as species in the making; and studied, not merely in their relation to disinfectants and "soluble ferments" (mere dead matter), but in connexion with their influence upon, and extent of being influenced by, far more minute forms of life than have hitherto been recognized as having a part to play in the world. It is at once apparent that a micro-organism can lose an "enzyme" or "enzymes" and acquire another or others. The ability to manifest hitherto unrevealed ferment powers can be acquired. The enzyme may, perhaps, be held, at any rate in some cases, to be a parasite rather than an organ. It is clearly, however, able to maintain its existence in the organism in an inactive or quiescent form.

The typhoid, paratyphoid, Gaertner, Aertrycke, &c., &c., organisms present every nuance within the range of what some would regard as the *species*, some as the *family* of organisms; and, indeed, it has been maintained that they overflow lines of demarcation so as to make clean-cut distinctions between the various types a matter of practical impossibility. The experimental results, in fact, suggest that each germ must be assigned a very complex constitution *qua* enzymes; one enzyme may be represented by numerous particles in a



condition of great activity and apt for rapid multiplication; another may be poorly represented by particles which are in an inactive or dormant condition.

The expression for such an organism may be given (with an attempt at mathematical exactitude) on the lines first indicated by Durham some twenty years ago as, say,  $1190a + b + 4c + 446d + \&c.$ :  $a$ ,  $b$ ,  $c$  and  $d$  being enzymes. A particular enzyme might (on this showing) rarely, if ever, be encountered *in puris naturalibus*; it would as a rule be associated with other ultramicroscopic, or it might be with actual visible organisms. If only an enzyme " $a$ " could be isolated and grown by itself upon the appropriate "substrate," how different the conditions from those of an experiment in which the said enzyme " $a$ " is multiplying in a Vanity Fair of an organism, so to speak, having, at a particular moment, say, the formula given above; and, multiplying moreover, under conditions of growth, not on a "substrate" of nicely defined chemical composition, but in a medium containing an olla podrida of proteids, carbohydrates, extractives, salts—to say nothing of vitamins, aggressins and the like—in varying proportions; indeed, it may be that the environment is not even a dead one, but that there actually lurk in it, unrecognized, numbers of living organisms. Under such conditions the interpretation of results of experiment must be difficult, and yet investigators, thus handicapped, have deduced the presence of "co-enzymes," "anti-enzymes," and so on. They may be right, at any rate no one can say they are wrong. From our new point of view there springs up, however, a profound scepticism as regards physico-chemical explanations of enzyme activity. The physico-chemist of course rests satisfied with his assumption that an enzyme is explained by calling it a "catalyst," though as Sir William Bayliss carefully points out: "At the same time the majority of them have certain incidental properties which distinguish them from the majority of inorganic catalysts, but this is all that we are justified in asserting."

This view does not throw much light on the possibility that some so-called "enzymes" may be living organisms, and it seems desirable to pursue the matter further. In the "Principles of General Physiology," Sir William Bayliss has fully set out the orthodox doctrine (p. 329). Fortunately, however, he has worked the whole thing out also, stage by stage, in the successive editions of his book, "The Nature of Enzyme Action." He has compared the various activities grouped under the head of "enzymic" with those known as "catalytic"; and, he argues, "If it is found that their nature is the same, an explanation has been given." Those of his readers who realize that he has "not yet fully explained the nature of enzyme activity" must appreciate that there is much virtue in that "if."

Close study of the "Nature of Enzyme Action" might, indeed, be held to afford more support to the hypothesis that enzymes *do not*, than to one that they *do*, "obey the usual laws of catalytic phenomena." To begin with, there is the fact that, while the true catalyst merely changes the rate of a reaction already in progress, it has to be conceded that in dealing with enzymes "a reaction does sometimes seem to be initiated." Further, the final result should not depend upon the amount of catalyst added; and yet "such cases are not infrequent among enzyme actions; and will be considered later." Moreover, "catalysts as a rule are definite chemical individuals of known composition and properties." . . . But, we read, "As yet this statement cannot be made of any enzyme. . . . We are not, however, warranted in denying chemical constitution to this class of bodies, until it has been shown

that bodies of known constitution may at one time possess the properties of enzymes and at another time, without any change in their chemical nature, be devoid of such properties." Here, it must be admitted, the *onus probandi* is skilfully transferred to the shoulders of critics and objectors. A little later it is agreed that practically the only "two properties common to all catalysts, viz., that of not initiating a new reaction, but merely changing the rate of one already in progress, and that of not appearing in the final products of the reaction, are both not infrequently not exhibited by enzymes." It is, thereupon, concluded "were it not that it seems impossible to place enzymes in any other category than that of catalysts, the anomalies above touched upon would be more serious"; but, it is added, perhaps, "on the whole, considering how little we know as yet about the intimate nature of catalytic phenomena in general, there is no doubt that the difficulty referred to will sooner or later be removed."

A great distinction between inorganic catalysts and enzymes is that the latter are destroyed by temperatures of from 60° to 100° C. This behaviour, "though of practical importance," does not, we are assured, "affect their (the enzymes') claim to be regarded as catalysts, and will be explained in the next chapter." There the attempt is made to solve the mystery. Enzymes are colloids, and carry down with them "by adsorption, constituents of the solutions from which they are precipitated." Hence, we are told, the query may be raised whether "enzymes, if obtained free from coagulable impurity, might not be found less sensitive to heat"? The heat test at any rate cannot, it is agreed, be relied upon in deciding as to the nature of a supposed enzyme. The "catalytic properties" are in fact "the only satisfactory means of solving the question." At this juncture, however, it is remarked that De Jäger and Arthus consider that "*various kinds of bodies may have conferred upon them the properties which cause them to behave like enzymes; so that we have to deal with properties rather than substances.*" Now we seem to scent the morning air and to ascend to a higher region of speculation than before, but a few paragraphs later we execute a *vol plané* and are near the earth again, for we read, "As already pointed out, inorganic catalysts are definite chemical individuals; there is, therefore, justification for holding that organic catalysts are also bodies of definite chemical composition, at all events until stronger evidence has been brought to the contrary."

Again, as regards the heat difficulty, we are again reminded, "This property is no doubt due to the colloidal nature of these bodies, and is in practice a useful means of detecting whether a substance belongs to this class of bodies or not." So that we are asked to accept a physico-chemical status for enzymes and if the objection be raised that in this guise they are just wolves in sheep's clothing, the reply is—"Just so, that is the beauty of it, not only have we here an instance of the exception proving the rule, but the fact that enzymes are peculiar in this particular respect enables us to determine whether we are dealing with the enzyme-catalyst class of bodies or not." There follows the candid admission that: "This characteristic of enzymes obviously gives us no help in deciding whether an action is due to an enzyme or to the agency of living cells. Perhaps the distinction is one of words only, but at present there are many changes known to be produced by living cells, changes of a kind such as no enzymes yet known are able to effect." (If this line of argument were further pursued, it might be concluded perhaps, that because an amoeba cannot play upon the piano it cannot be held to be alive.)

The antiseptic test, it is pointed out, is in even worse case than the heat

test, for while protoplasm is, as a rule, injured by an amount of antiseptic which has little or no effect on enzymes, "some enzymes are very sensitive to certain antiseptics, such as formaldehyde." Filtration through porous clay or Berkefeld filters seems often to exclude living cells but cannot be implicitly relied upon. Next we voyage over the dark waters of "reversible actions" and "velocity of reaction," and reach the most serious difficulty of all from the mere physico-chemical point of view, that of "optimum temperature." Duclaux held this was "evidence of vital action"—but no, there is again an appeal to the "colloidal condition" . . . "this with its sensitiveness to heat is the cause of the phenomenon in question." A further reference to antiseptics and to the difficulty of the "very specific nature of certain enzymes," concludes the case for (or against) regarding enzymes as catalysts. It would simplify things a great deal if, taking our courage in both hands, we boldly declared that many "organic catalysts" are really living organisms.

In the third edition of "The Nature of Enzyme Action" there is some modification of the original position—a withdrawal as it were to an inner line of defence. Enzymes, we are now told, are not proteids. The specificity doctrine is less esteemed than heretofore, and the lock and key simile, it is said, has been overworked. "The subject needs attacking from the dynamic and not merely from the static point of view." "Locks do not open themselves, however long a time be allowed for the process to take place." "Mere structural chemistry offers very little help in a difficult problem." "We must give up specificity except in a very wide sense." Brailsford Robertson's note that "adsorption as a physical process has a low temperature co-efficient. whereas enzyme action, as a whole, has the high co-efficient of a true chemical reaction," does not fit in with these modified views; chemistry, however, is (in this third edition) out of favour and physics may be said to be paramount. Close inspection of the new front suggests that the defences would be greatly strengthened if it were insisted upon that something dynamic, not static, operated on the adsorption compounds at the interfaces between the phases—in fact that the submicrons and microns there may in some instances be held to be alive.

It is refreshing to escape from this close air, in which we are "seeking a way and finding not the way, but toiling desperately to find it out," to the calm atmosphere of the address of the late Professor Minchin, delivered in 1912, and entitled "Speculations with regard to the Simplest Forms of Life and their Origin on the Earth."<sup>1</sup> This address goes far to support the claim that ultravisible particles may be living organisms. Dr. Minchin first refers to the fact that in the lowliest forms the most distinctive property of living things is that known as metabolism. "Could we either magnify the substance of the living fabric, or increase the range of perception of our senses to such an extent that we could strictly observe the chemical and physical changes taking place, then a living organism, however minute, would appear to us to hum like a factory or roar like a furnace."

He then gives a remarkable account of the functions of the "chromatin grains" in cytoplasm, and refers to their relation to ferments or enzymes, "substances which, more than any others, are characteristic of living bodies and of vital activities." He declares that if chromatin should prove unable to live when removed from its natural surroundings, this would not show that it was not "the primary living substance, any more than the fact that a fish cannot live out of water would be a proof that the water was living as well

<sup>1</sup> See *Science Progress*, 1912-13, vii, p. 300.

as the fish." He states that many organisms "appear to consist mainly, if not entirely, of chromatin alone, as for example some bacteria and spirochaetes, and above all the organisms known as Chlamydozoa." The last named are so small that they pass filters, and yet they are known to be independent living forms, playing, it is now believed, an important rôle in connexion "with certain very well known diseases of man and animals." Minchin instances small-pox, vaccinia, trachoma and molluscum contagiosum in human beings; and in birds, epithelioma contagiosum and diphtheria"; he further hints at other possibilities. He says "the primary living substance, the *primum vivens*, is chromatin; and from that I draw the conclusion that the simplest and earliest forms of life were minute particles of chromatin, without other structural accessories, but nevertheless capable of the essential and characteristic activities of living things. That is to say, of assimilation, growth and reproduction by fission." The most primitive type, he holds then, is "not a relatively large cytoplasmic organism, but an extremely minute body, a tiny speck of chromatin." He adds that, "if any organisms exist at the present day which represent the original type of living being in its primitive form unchanged through the ages . . . such organisms would have to be sought among parasitic or saprophytic organisms; that is to say, obtaining their supply of organic matter either from a living body or from one that has lost its life recently." Truly, Professor Minchin proffered here to Sir William Bayliss a world of ultravisible living particles with which he could people his interfaces and stimulate dynamical activity.

Dr. Minchin himself held that "life carries on its characteristic activities subject to and restrained by the physico-chemical laws of matter, but does not owe its origin to those laws, and is not, perhaps, in other worlds, bound up with the same forms of matter with which it is connected in ours. The minute chromatin particle or germ of life, he said, might conceivably, on another planet, set in motion vortices of metabolic change quite different from that type with which we are acquainted here."

We may then trace the following stages in the evolution of accepted doctrine with respect to fermentation. First there is the stage when, as Lister described it, a distinction was drawn between the living organism, capable of reproducing itself and of exhibiting ferment properties, and the unorganized chemical ferment (emulsin, ptyalin, pepsin, are the examples he instanced).<sup>1</sup> Then came the differentiation effected by de Schweinitz and Buchner, between the germ (bacillus or yeast) and the ultravisible virus or zymase associated with it. Dr. Vernon placed the enzyme "secreted by living substance" in an intermediate position "between the ordinary protein molecule and the living organism." Bayliss lays stress on the physical properties of colloids, and does not expressly affirm that any enzyme is a living organism. Minchin, however, goes much further than this. Reiner-Müller, Massini, and others show that differing cultural appearances result when bacteria undergo mutation, i.e., when they acquire new fermentative

<sup>1</sup> The position reached in the early nineties may be illustrated by reference to the following inquiries. Dr. Copeman had reported (International Congress of Hygiene, 1891), on a special method for the "bacteriological purification and for the preservation of vaccine lymph." This method involved separation of the vaccine virus from associated contaminating germs. Looking at a similar problem from a different angle and working in Dr. Klein's laboratory, I was able "to obtain material which is capable of manifesting ferment powers altogether apart from the growth and development in it of micro-organisms" (Local Government Board, Annual Report, 1891-92, Appendix No. 5). The Medical Officer of the Board commenting upon this says "the experience . . . may prove of service in the study of the action of unorganized as contrasted with that of organized ferments."

properties. Then comes a further development which must now be referred to. Twort, in 1915, in "an investigation on the nature of ultra-microscopic viruses," described a peculiar change occurring in colonies of micrococci cultured from vaccine virus. He speaks of the change as possibly being due to "a disease of the micrococcus." In 1917 d'Herelle gave an account of his study of a living filterable organism parasitic on bacteria, obtained from intestinal contents in cases of dysentery, and which he termed a "microbe bactériophage." The phenomenon under observation is pretty generally believed to be one and the same, in both instances, and is often spoken of as the "Twort-d'Herelle" phenomenon. d'Herelle lays stress on the fact that, by adding a few drops of a Berkefeld filtrate from one of his cultures, an active sub-culture capable of effecting lysis of the bacteria is obtained: and this sub-culturing procedure can be repeated indefinitely, thus giving an impression of growth of a parasite. Bordet (1921), on the other hand, maintains that the active agent is an autolytic enzyme produced by the bacteria. Thus, while d'Herelle, in this modern conflict, argues, as did Pasteur fifty years ago, that a living germ is at work, Bordet, Pasteur's great follower, takes up a position more similar to that held in the great controversy of "the seventies" by Liebig. d'Herelle is for an "ultravisible parasite," Bordet for an "autolytic principle." Parasite or principle, which you will, is capable of growing in (or with) the germ, and of bringing about vitiation of the bacterial metabolism resulting in lysis. According to d'Herelle there is multiplication of the agent causing the lysis, hence it is living. Bordet does not question the increase in amount of the principle, but denies that there is growth of a living germ. He appeals to the analogy of the production of more thrombin, when a trace of thrombin is added to uncoagulated plasma. He admits, however, that this analogy is not an altogether satisfactory one. He seems to hold that the thrombin does not reproduce itself, whereas his "principle" does. Inasmuch, however, as the plasma must, for the purposes of the experiment be living plasma, it might be contended that it really constitutes a reservoir of the living precursor of thrombin. So that Bordet insists that the growth of his principle is only possible when it has at command a supply of the appropriate living protoplasm. While he demurs to accepting the designation *parasite*, it is not clear that he would not be prepared to accord to his principle the status of an *organ*.

An outstanding difficulty of the epidemiologist, confronted with all the later developments of the germ theory, is, however, that of explaining *persistence of type* in epidemic disease—persistence such as that displayed, say, by measles or small-pox; or (when multi-annual waves are smoothed away by taking a long view over two or three decades) in scarlet fever, diphtheria and influenza. True, geology tells us that "the most lowly organisms, provided their environment remains an unchanging one, are the most enduring" (Geikie); and, in the case of ultravisible germs of epidemic disease, it may be that, while variability of type is favoured by association of the primary ultravisible organism with various satellite secondary invaders, there is an inevitable return, at appropriate intervals, to phases of influence which again and again repeat themselves. Just as syngamy prevents a species from breaking up into strains, so some equilibration between a primary influence and secondary influences encountered in the bodies of the hosts, may bring about major waves of epidemic disease. De Vries has hinted at something of this kind in his "periodic mutations" of the higher plants, and analogous phenomena will at once suggest themselves to all those who have followed up Creighton's teaching with regard to influenza, scarlet fever and diphtheria. Minchin's ultramicroscopic chromatin grains



may then become endowed by mutation with the capacity for exercising marvellous powers; for while an evening primrose, when it mutates, causes, comparatively speaking, but little disturbance in the world at large, the ultra-visible germ of influenza, on the other hand, when it manifests its greatest effects, the pawn becoming (so to speak) a queen, determines development of influenza in pandemic phase, "posting" abroad and wreaking terrible havoc among the peoples of the globe. The mutation periods presumably represent on this showing the result of interplay between germ and increasingly or decreasingly immunized environment.

The lines of argument which have been followed seem to converge and concentrate upon a point, and the attempt must be briefly made to focus them. To begin with, as Sir William Bayliss noted, at the beginning of his book on "Enzymes," a distinction must be made between "trigger action," and the more obtrusive energy produced by the explosive material with which the gun is loaded. Sir William Tilden, in "Chemical Discovery and Adventure," says in his chapter on "Enzymes," that the important question is the extent to which "the processes of absorption and assimilation, of growth and development, are wholly dependent on catalytic processes, or are at least partly the result of complex changes wrought by the living protoplasm itself." The battle ground of "the seventies" in which Liebig, Pasteur, Tyndall and Bastian played such notable parts is thus once more the scene of conflict, but the fight now concerns the vile bodies of organisms far smaller than those about which the archebiosis and spontaneous generation controversies raged. There is, partly on this account, more than ever a tendency to maintain that ferments are grossly material, and there are even those who think that the Emil Fischers of the future will be able to synthesize living organisms.

But mere mechanism will never do. As Sydenham said 250 years ago "it is in accordance with immutable laws, and by a scheme known to herself only, that parent Nature accomplishes the generation of things." Sydenham's friend, Robert Boyle, moreover, held that "a fair account of fevers would never be given without an insight into the doctrine of fermentation." He believed, too, that the same matter may cause divers diseases, and that "by metastasis of the morbid matter (as for instance how that which in the lungs caused a violent cough, removed up to the head, may produce a quick decay of memory and ratiocination, and a palsie in the hands and other limbs"). . . . Then, inspired, doubtless, by his friend Sydenham, he continued . . . "Diseases that appear very differing, may easily be produced by a peccant matter of the same nature, only variously determined in its operations by the constitution of the parts of the body where it settleth." A somewhat similar view seems to have been adumbrated by Willis, and was held two centuries and a half later by Creighton; it has been examined in recent years in papers and addresses by various members of this Section; it has lately been glanced at with some measure of approval by Dr. A. J. Hall, in his recent Lumleian Lectures; it has also quite recently found expression in a Report to the Medical Research Council.

In this last-named instance the writer, Surgeon-Commander S. F. Dudley, "fully recognizes the accuracy and importance of Sydenham's classical observations upon change of type in epidemic disease and the value of similar material collected by later clinicians and epidemiologists." But he does not think it necessary to invoke "mystic unknown factors." "All the peculiarities of epidemics," he says, "will be ultimately explicable, on well known biological first principles, as due to the constantly changing relations between man and his parasites." We must, however, include the ultraviolet as well as the

visible germs in the scope of the inquiry. Sydenham's doctrine seems, in fact, to be now entering upon the third stage in the development of new conceptions. "First," so we are told by William James, "a new idea is absurd, then trivial, then begins the chorus, 'we discovered it.'"

The doctrine comes into line, moreover, with other modern ideas. Rather more than half a century ago, following upon advance made in the biology of the infinitely little, Darwin, in propounding his hypothesis of pangenesis, urged that "an organic being is a microcosm, a little universe formed of a host of self-propagating organisms, inconceivably minute, and numerous as the stars of heaven." In quite recent times Haldane has insisted upon the insufficiency of mechanism on the one hand, and of vitalism on the other, and claims that what he terms "organicism" is the factor to be reckoned with. He affirms that "the spiritual interpretation, as the supreme interpretation of the universe is coming again into its own." And, he says, "we cannot either in deed or thought separate living structure from its environment. We cannot add up in a sum units which do not exist separately. Hence the life of a compound organism is no mere sum of the lives of its constituent cells." Again, the development of knowledge concerning parasitism in very lowly forms of life must be emphasized; also the hypothesis already outlined many years ago by Dr. Ross in his "Graft Theory of Disease," and the recent observations of Keeble; these all have a close bearing upon the questions at issue. Finally the mutation theory of De Vries: its application by latter day biologists and bacteriologists; and its suggestiveness, particularly when we remember De Vries' views, as regards "periodic mutation"; these all have to be considered in relation to Sydenham's hypothesis of epidemic constitutions, to his observations concerning intermittent fevers, continued fevers, new fevers and so forth, and to Creighton's much later speculations regarding agues and influenzas.

Huxley said that it was the business of the inquirer "to follow humbly to whatever abysses Nature leads." But how difficult is the exploration of life at these abysmal depths. The astronomical enthusiast in "Two in a Tower," speaks of the degrees of *immensity* revealed in stellar space; and Thomas Hardy makes him describe one after another the successive vastnesses of "dignity," followed by "grandeur," "solemnity," "awfulness," and an ultimate "ghastliness." Proceeding quite contrariwise, so to speak, we may traverse abysses, not of *immensity*, but of *minuteness*, travelling first through the domain of "lowliness" (at  $10^{-4}$  cm.) to reach in turn "insignificance," "ultravisibility," then the meticulous "awfulness" of the particles of Sydenham and Boyle, and ultimately the final stage, "molecular dimensions" ( $10^{-8}$  cm.). The claim that strict limitation must be placed upon the scope of epidemiology is perhaps more insistent now than ever before, and the epidemiologist of to-day is only too apt to fall into the mood described by Shakespeare:—

In disgrace with fortune and men's eyes,  
He all alone beweept his outcast state . . .  
Wishing him like to one more rich in hope . . .  
Desiring this man's art and that man's scope,  
With what he most enjoys contented least . . .

and yet, when "in these thoughts himself almost despising," haply he thinks of the teaching of his master Sydenham, and that, remembered, "such wealth brings" that he scorns to change his state even with the mechanical philosophers.



## DISCUSSION.

Dr. J. A. ARKWRIGHT said that he was more interested and concerned with what Sir William Hamer had to say about recent advances in biological knowledge and their bearing on the causation, prevention, course and treatment of infective disease than with the suggestions put forward by Sydenham about "epidemic constitution," which he (Dr. Arkwright) neither disputed nor accepted. Dr. Arkwright said that the bacteriologist either was, or else strove to be, a collector of facts such as he could observe or elicit by experiment, and that he made use of these facts to test the various hypotheses which came from the fertile brains of philosophers of all ages and were often reproduced with slight changes again and again. He pointed out that these hypotheses had to be specially adapted to each individual subject of inquiry, and that most of them had to be discarded as incongruent with the facts, whilst a few were kept and were further tested. He maintained that it was the new facts which were of value, for they enabled the seeker after truth to swim in the ocean of fancies which threatened to drown his reason and overwhelm his mind. Sir William Hamer had, if he (Dr. Arkwright) understood him rightly, criticized the attitude of bacteriologists, or rather students of pathogenic microbes, especially on two or three points:

(1) He said that they took too crudely physicochemical a view of biological activities and he particularly blamed them for discoursing of enzymes of which they knew little. Dr. Arkwright said he was not aware that any student of biology took the enzyme doctrine light-heartedly, but that the phenomena were definite enough, and in default of being able to see and feel these agents, it was surely excusable to infer their existence from their activities, a course which had to be pursued in dealing with far more tangible matters.

(2) Sir William Hamer rallied them with beating about the bush and debating on the nature of ultramicroscopic viruses instead of "plumping" roundly for a living body and substance as their essence. He (Dr. Arkwright) was not sure whether Sir William Hamer did not intend this latter criticism sarcastically. Taken seriously it came rather as a shock from one who progressed so cautiously as Sir William. To more pedestrian minds the question seemed a hard one to settle outright in some instances. For example at the present time there was much keen research into the problem of the Twort-d'Herelle phenomenon. The facts were accumulating, but there was not enough evidence either way completely to satisfy many of the inquirers. There were protagonists on both sides who after weighing the same evidence could do no more than arrive at a probability in favour of one side or the other. The one side inclined to a living, the other to a dead active principle existing in a highly efficient liquid which after being filtered contained no visible or cultivable bacteria, and might be subjected to the action of chloroform water, 2½ per cent. carbolic, or to a temperature of 56° to 60° C. for an hour and yet still contained the active, transmissible lytic agent. He (Dr. Arkwright) claimed that surely in this matter bacteriologists could not be accused of laziness, supineness, or undue adherence to old formulae. He maintained that this inquiry, if steadfastly pursued, might yet lead to a definite solution, and indeed might add one more link to those connecting living and dead matter. He remarked that it had been pointed out more than once lately that phenomena resembling life or spirit so pervaded matter that it might be truer to say that all matter was imbued with life, than that living things were merely a complicated arrangement of dead matter.

(3) Dr. Arkwright thought that Sir William Hamer's more intimate and serious criticism was concerned with the problems of change of type of disease which were put forward by Sydenham, and with the difficulty which bacteriologists had encountered in establishing the occurrence of variation or mutation of pathogenic bacteria. It was not necessary to point out to Sir William Hamer that variation in the causal micro-organism was not the same thing as variation in the disease. As far as he (Dr. Arkwright) knew, there was no definite evidence that the pathogenic organism of one disease ever changed into another which caused another different disease—presuming the ordinary ideas of the boundaries of diseases to be accepted. He believed, however, that after much careful work it had been established that certain pathogenic bacteria did vary in culture very much as regards degree of virulence and certain other

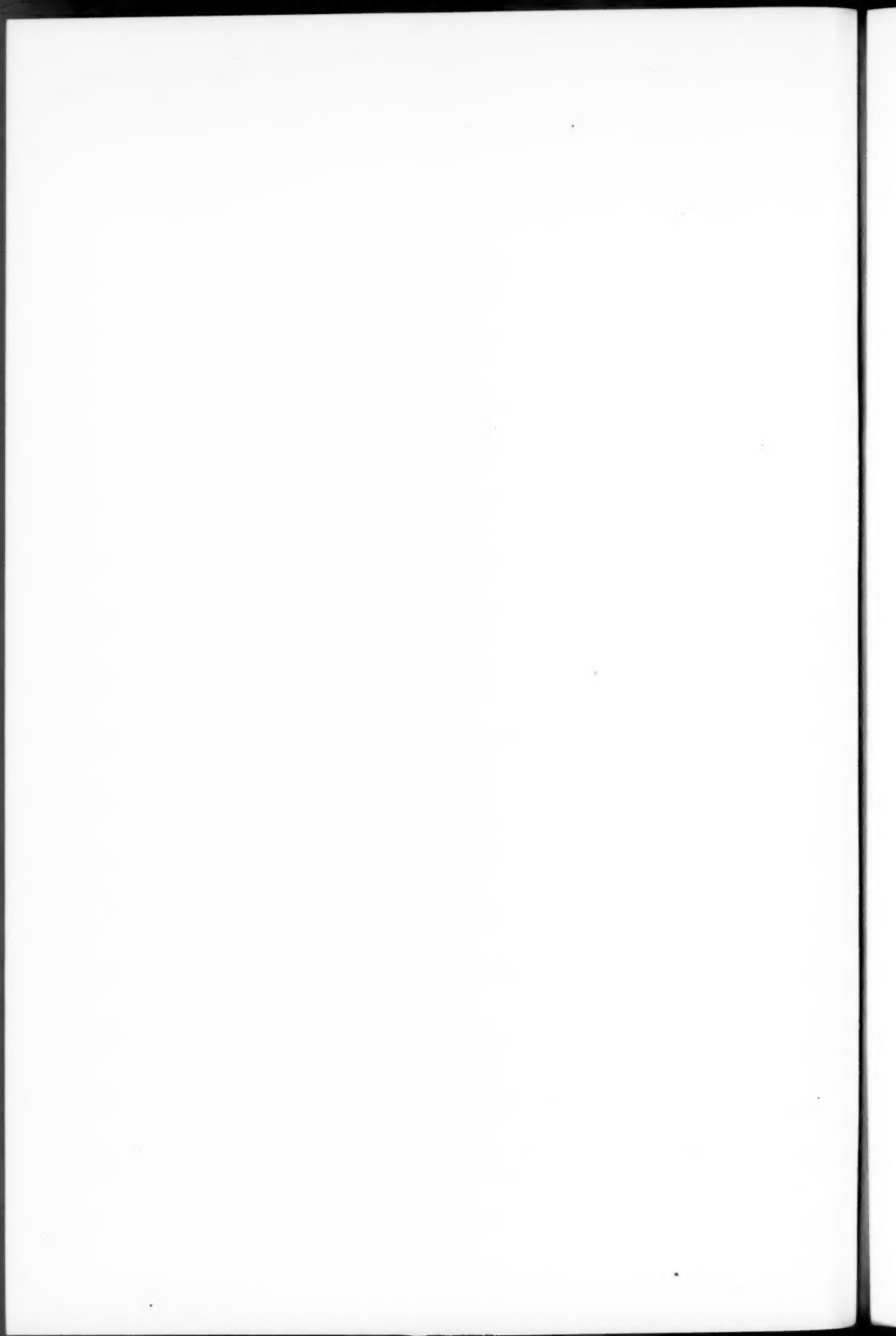
properties, excluding, however, changes relating to the kind of disease which they were capable of producing. It seemed probable, indeed, that such a change as those last mentioned could only take place in the complicated environment found inside the body of the host, and that such sound evidence as had been so far available had not, he believed, pointed to such an occurrence. There appeared to be a great stability in bacteria in certain characters, as in other living things. It was quite obvious that in dealing with such small objects, which it was so difficult to characterize and recognize, it had been very important to establish the type before studying the variants. He claimed that the large amount of work that had been done on variation had led to greatly increased knowledge and that there was still a large and important field being explored with a view to discovering how far, how often, and under what conditions bacteria did vary and to what extent this could be controlled. He remarked that evidently Sir William Hamer had begun to hope that bacteriology would confirm Sydenham's notions, but the study of facts was a slow and laborious process and did not always lead in the same direction as the prognostications of the ablest prophets.

Dr. S. MONCKTON COPEMAN, F.R.S., said that Sir William Hamer's paper formed an admirable *résumé*, to date, of existing knowledge on the subject of what were known as ultravisible viruses, and as such, would be most helpful to those who might not have the opportunity of themselves consulting the numerous sources of information laid under contribution. At the same time, however, it did not necessarily follow that one was in agreement with all Sir William Hamer's conclusions and inferences. There was still much to be learnt as to the biological characteristics of these extraordinarily minute bodies, and more especially as to the methods most likely to be productive of success in their cultivation. Further knowledge also as to their symbiotic inter-relations with bacteria was essential. It was not improbable that the outcome of team research work on a large scale, and with the aid of ample funds, on the disease of dogs known as distemper, which was about to be initiated under the direction of a committee of scientific experts, might prove fruitful in all these directions, as the main idea of the research in question was the study of a supposed specific ultravisible virus. The speaker, many years ago, had isolated from dogs suffering from, or dying of, this disease, a minute coccobacillus which appeared to be capable of giving rise to attack by the disease when inoculated into a healthy dog. But it was possible that, as in the well-known case of hog-cholera, an ultravisible virus might also be concerned in the matter. Should this prove to be the case, the conditions afforded by the special circumstances under which it would be possible to carry out the investigation could hardly fail to add largely to our knowledge of the bionomics and epidemiology of ultravisible viruses.

Dr. R. DUDFIELD said that it was presumption on his part to intervene in the discussion owing to the fact that he was not educated in the science of bacteriology. That science was unborn when he was a student, and he had had no opportunity by reason of official duties to make good the defect. His desire was to find out why a disease, such as diphtheria, was now sporadic and now epidemic. He had chosen that disease because he knew something—a very small something—about its bacteriology. He thought that three and possibly four varieties, or developmental stages of the Klebs-Loeffler bacillus existed, viz., the "Hoffmann," the "a-virulent," the "virulent," and the "epidemic." They were all familiar with the fact that an infinite number of generations were produced in a finite space of time. He was inclined to the view that the Klebs-Loeffler bacillus was capable, in suitable environment, of passing up and down the scale represented by the four "varieties" mentioned. When the disease was sporadic the only factors to be considered were those of host (the patient) and bacillus. Given the possibility of mutation, he could conceive the possibility of the bacillus acquiring, in its passage through various hosts, the quality of "epidemicity." Would it be beyond reason to imagine that some reaction similar to the Twort-d'Herelle was the cause of the new quality in the bacillus? Might not the bacillus itself be "sick," and hence specially capable of causing disease? Sir William Hamer had referred to Sydenham's hypothesis of "epidemic constitution," but no one, apparently, knew exactly what that hypothesis connoted. If his (Dr. Dudfield's) speculations were worth anything, might

not "epidemic constitution" be taken to mean the changes in the bacillus itself—doubtless the result of many factors?

Sir WILLIAM HAMER (in reply) said that there was, it had been said, a doubt as to whether Sydenham really knew what he meant by an "epidemic constitution." Some critics did not like "skiey influences," others the references to the "bowels of the earth," and it was certainly difficult in these days to contemplate the existence of a variola without spots, though in very exceptional cases there was a near approach to this state of things. But after all these were only some of the trees, and they must not be allowed to obscure the fact that there was a wood. Sydenham held that one and the same "fever" might present at one time one set of symptoms, and at another time another set of symptoms possibly of quite a differing character. Every epidemiologist who, after study of Sydenham, Willis and Creighton, had given any consideration to the question, appreciated that this fact was one of the corner stones of the new epidemiology. Sydenham's words, it had further been urged, seemed to imply a belief in "mystic unknown factors." But how could Sydenham possibly have had complete foreknowledge of the work of Pasteur, Lister, Buchner, de Vries, Creighton, and modern laboratory workers concerned with ultravisible viruses? As was explained in Sheridan's "Critic," one could not see the Spanish fleet if it was not yet in sight. In the same way Lister was at a disadvantage, in 1878, in that he had not before him such results as those of Buchner and Keeble. Had he been familiar with the facts relating to symbiosis, which were now generally recognized, he would doubtless have applied them to the problem he was considering. It was of course up to the student of epidemiology to avoid any materialistic bias, and to pay attention to notions as well as to sense-experiences.



## Section of Epidemiology and State Medicine.

President—Dr. R. J. REECE, C.B.

### The Incidence of Venereal Disease in Scotland.

By T. F. DEWAR, C.B., M.D., D.Sc.

(*Medical Officer, Scottish Board of Health.*)

(ABSTRACT.)<sup>1</sup>

DR. DEWAR'S paper was an endeavour—by means of a survey of all the results of work that had been published in connexion with the incidence of venereal disease in Scotland, especially as demonstrated by series of Wassermann tests—to attain a fairly reliable conception of the general incidence of syphilis and gonorrhœa among the population of Scotland. He explained that the work had arisen owing to a realization of the necessity of convincing members of Scottish local authorities that these diseases were by no means exceptional in at least the populous parts of the country. Dr. Dewar admitted that in any inferences that could be arrived at regarding the incidence of venereal disease in Scotland there must be a large element of conjecture, and that such incidence undoubtedly varied very much between the cities on the one hand and the sparsely populated areas, the islands and peninsulas of the north and west, on the other.

Dr. Dewar discussed at some length the incidence of illegitimacy in Scotland, which is high on the whole, and strikingly different as between one area and another, certain counties maintaining year by year a pre-eminence in this respect. He considered it as at least probable that the venereal disease incidence-rate varies from place to place in approximately inverse proportion to the illegitimate birth-rate. Admitting that there was particular difficulty in estimating the incidence of venereal disease in the rural and mountainous districts, he pointed out that in respect of 80 per cent. of the population of Scotland, who were congregated in the Forth and Clyde basins and in the immediately adjoining mining and manufacturing districts—i.e., four-fifths of the people being resident in one-eighth of the area—the incidence of venereal disease was not likely to vary greatly.

Passing to the statistical material at his disposal Dr. Dewar first dealt with the figures available in the Registrar-General's reports, pointing out that little reliability could be placed upon the number of deaths attributed to syphilis, since about 75 per cent. of these were of children under 1 year old. In other words, whilst congenital syphilis is certified as a cause of death with some candour, at least in infants, acquired syphilis is quite exceptionally given as a cause of death. He quoted statistics and estimates collected by Dr. Dittmar, of the Scottish Board of Health, in 1919; these figures, based upon the death returns, assuming that all cases of general paralysis and locomotor ataxia are definitely syphilitic and that other diseases such as aneurysm, hemiplegia under

<sup>1</sup> This paper will be published in full in an early issue of the *Edinburgh Medical Journal*.

55 years of age, and "congenital debility" are attributable to syphilis in a fraction of all the cases which can be approximately measured. On this basis Dr. Dittmar concluded that in Scotland on an average about 4,200 persons, or 92 per 100,000 of the whole population, die of syphilis; this figure being equivalent to just under 6 per cent. of the total annual death-rate. It is significant that Dr. David Watson, Glasgow, who has had very wide experience of syphilis in Scotland, has expressed the opinion that Dr. Dittmar's estimates are too low. He formed the estimate in 1919 that in Scotland annually there were about 27,000 new cases of syphilis and 85,000 new cases of gonorrhœa. It is significant to compare this estimate with that given by Dr. Douglas White in his evidence before the Royal Commission on Venereal Diseases, who, also basing his estimate upon a consideration of all available statistics, came to the conclusion that in the United Kingdom there were annually about 114,000 fresh cases of syphilis and about 686,000 fresh cases of gonorrhœa. If the latter estimates are correct, then either Dr. Watson's figures are too high in respect of syphilis, or Scotland has a higher incidence of that disease than England. As regards gonorrhœa, the two estimates are in surprisingly close agreement.

Dr. Dewar thought that a more accurate estimate was to be attained by study of the numbers of deaths attributable to general paralysis and locomotor ataxia—both undoubtedly syphilitic in origin. Upon a review of all the figures available as regards the percentage of cases of syphilis that ultimately manifested one or other of these sequelæ, and especially of the exhaustive investigation of Mattauschek and Pilcz, who traced the course of syphilitic illness in over 4,000 officers of the Austrian army, it seems fair to conclude that, taking both sexes and both end forms of the disease, from 2 to 3 per cent. of all the persons who acquire syphilis eventually die of general paralysis or tabes dorsalis. Applying that factor to the average number of deaths annually ascribed in Scotland to these maladies, it appears that at a period about twelve or fifteen years previously, from 8,000 to 13,000 persons annually acquired syphilis in Scotland. If 10,000 is taken as a provisional figure, it is not likely that it will be far from the truth.

The most reliable basis for an estimate of the incidence of syphilis in Scotland is undoubtedly to be derived from the records of the application of the Wassermann test to various groups in the community. Dr. Dewar quoted the statistical results of a large number of such investigations in Scotland. He quoted Dr. W. M. Elliott, who examined the blood of 130 children admitted to measles and whooping-cough wards in Glasgow: out of 126 of these who presented no indications of syphilis, ten gave a positive reaction; Dr. Ivy Mackenzie, who found that of 786 blood samples from insane patients, 347 gave a positive reaction; Dr. Thomson, Medical Officer of Barnhill Poorhouse, who found that of 81,244 patients consecutively admitted, 1,955 suffered from venereal disease of acute stage or type; Dr. Kennedy, Maternity Hospital, Glasgow, who found that of 1,881 consecutive samples of maternal blood examined, 9.03 per cent. were positive; and that of 1,350 samples of blood taken from infants, 4.14 per cent. were positive; Dr. J. N. Cruickshank, Glasgow, who, among 1,900 unselected samples of maternal blood, found a positive Wassermann reaction in a little over 9 per cent.; Dr. M'Iroy and Dr. H. F. Watson, who took samples from 100 unselected out-patients at the gynæcological clinic of the Royal Infirmary, Glasgow, finding 43 per cent. positive; Dr. Kate Fraser and Dr. H. F. Watson, who found 46 per cent. of positive Wassermann reactions out of 204 cases of abnormal children attending special schools in Glasgow;

Dr. Carl Browning, who mentions 331 unselected cases of children attending Glasgow dispensaries, of whom 14 per cent. presented evidence of syphilis; Dr. John Watson, who examined the blood serum of 619 patients at the Tuberculosis Hospital, Robroyston, Glasgow, finding 6.6 per cent. positive; Sir Norman Walker, who found that of sixty-five consecutive cases attending his lupus clinic at the Edinburgh Royal Infirmary, five were positive; Dr. E. M. Dunlop, who examined the blood of seventy-seven cases of scarlet fever admitted to the County Hospital, Motherwell, none of whom were found positive; Dr. W. T. Munro, who among 100 patients admitted to the Glenlomond Sanatorium, Fifeshire, found twenty positive; Dr. W. J. Tulloch, Dundee, who found that of 109 specimens sent from the Infant Hospital, Dundee, six were positive and two others doubtfully positive; and other authorities who had worked on similar lines and reached comparable figures.

Dr. Dewar mentioned that he had had a series of blood specimens taken from the operating table of a provincial hospital in Scotland; of the first ninety-eight, two were positive; while a second series now being examined shows four positive results in forty-three cases.

Dr. Dewar considered the evidence that was afforded by the number of attendances at the various venereal disease clinics in Scotland. These clinics number thirty-eight in all, of which thirty-three have outpatient departments. During the year ending May, 1922, 11,154 new cases had attended these clinics. He was not inclined to set much store by this figure, since the attendance at the various clinics seems to be more determined by the energy of the personnel than by any other local factor. Moreover, about one-half of the cases that attend are cases of syphilis. It is quite certain that the cases of gonorrhœa far exceed those of syphilis in actual number.

The inferences to be drawn from the report of the Ministry of National Service were also considered.

The question of the incidence of syphilis and gonorrhœa among troops and naval ratings in Scottish areas was considered somewhat fully, especially in its bearing upon the question of the general incidence of these diseases among the civil population.

The figures applicable to Scotland quoted in the report of the Royal Commission on Venereal Diseases were referred to, especially those regarding the incidence of the diseases among prisoners in Scotland.

The tentative estimates reached by all these diverse paths were reviewed in the light of the opinions held by those who had been associated with the treatment of venereal disease over considerable periods of time.

The author then dealt with the relative incidence of syphilis in the two sexes; with the comparative incidence of syphilis and gonorrhœa; and with the relation of the number of new cases occurring in a community in any one year to the total number of cases existing in the community at a given time.

#### CONCLUSIONS.

(1) It will be accepted by all that syphilis in Scotland has its maximum prevalence in Glasgow and the other large centres and that the incidence diminishes with the density of population until in the remote highland and insular parts it falls to a very low, if perhaps never quite trivial, rate.

(2) It seems to be the case that in the cities and large towns from 3 to 5 per cent. of the children are born with congenital syphilis. If the figures ascertained by Elliott perhaps point to too high an incidence, it seems probable that the estimate of Cruickshank is too low. It is significant that Dr. Leonard Findlay with his great experience of juvenile syphilis in Glasgow states—and



seems to accept the reproach—that “until the present time, with the exception of Epstein's Prague statistics, the highest figures have been obtained in Glasgow and have earned for it the unenviable reputation of being the most syphilized city in Britain.”

(3) As regards comparative incidence between Scotland and the remainder of Britain, there is good reason for accepting the dictum of the Royal Commission that “the general incidence of syphilis does not differ greatly in the two portions of the United Kingdom.”

(4) On a survey of all the statistics and estimates, it may be assumed that in Scotland, with a population of about four and three-quarter millions, 12,000 new cases of syphilis, on the very lowest computation, are annually acquired. Two-thirds of these are in men. Other observers make the total figure much higher, Dr. D. Watson making it fully twice as high.

(5) Each year, in Scotland, the new infections of gonorrhœa are from 35,000 to 70,000.

(6) At any given time, at the lowest estimate, 2 to 3 per cent. of the total population are infected by one or other of the venereal diseases. In the populous areas, the minimum figure is 5 per cent. for men and 2 per cent. for women.

(7) The tendency is certainly, if not in any marked degree, towards reduction of incidence of both diseases. This was to be anticipated on epidemiological grounds. It is well known that such diseases after being endemic for many years attain an approximate equilibrium. That being so, the work of the treatment centres must certainly have had the effect of temporarily, if not permanently, checking their prevalence.

#### DISCUSSION.

Dr. M. GREENWOOD said that Dr. Dewar's paper contained a valuable collection of statistical data which merited careful study. No department of medical statistics was more beset with pitfalls and it was necessary to exercise the greatest caution in drawing conclusions. From an instantaneous photograph—as it were—of the condition of a sample of the population, it was hard—in fact it was impossible—to deduce the information they would all like to have, viz., an answer to the question—What was the probability that a man (or woman) would contract syphilis before dying? Even the significance of the instantaneous photographs was hard to assess, as the variations in Dr. Dewar's series proved. These difficulties in no way detracted from the value of the impartially collected data which Dr. Dewar had brought to their notice.

Dr. R. DUDFIELD said that, bearing in mind the two-fold objects of the Section, he would deal with the prevention of venereal disease. As medical officer of health of a metropolitan borough having a population much greater than many county boroughs, he was debarred from taking any share in the treatment of venereal disease, the County Council being the authority for the metropolis. He had therefore no knowledge of the prevalence of the disease in his area, nor of the proportion of infected persons who ceased attendance at the clinics before completion of treatment. Although he understood that the records kept at such clinics made it possible for any given patient to be counted for statistical purposes two or more times, he regretted that so little use was made of the data available. Dr. Dewar's estimate of 12,000 new attacks per annum, with a ratio of three cases of gonorrhœa to one of syphilis, indicated a prevalence far in excess of those of any of the infectious diseases at present subject to measures of prevention. He (Dr. Dudfield) was satisfied that effective treatment ought to be established. While he feared that public opinion was not ripe for notification of every case of venereal disease, he thought that efforts should be made to secure “conditional” notification such as was in force in Western Australia and Canada. There could be no doubt that the spread of the disease was largely due to infected persons giving up treatment before cure.

## Section of Epidemiology and State Medicine.

President—Dr. R. J. REECE, C.B.

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### An Inquiry into the Mortality of Coal- and Metalliferous-miners in England and Wales.

By EDGAR L. COLLIS, M.D.

THE mining community is one of considerable interest. It comprises a compact body of workers who for the most part do not change their occupation. The work, whether concerned with getting coal, or mineral ores, has much in common; it is carried out underground, in excavated workings, dimly illuminated and artificially ventilated. Considerable physical exertion is called for, and there is exposure to dust of the materials worked. The workers are away from their homes for eight to nine hours a day and carry with them any food or drink they consume during working hours. Notwithstanding these similarities in their working life the mortality experienced by different groups of miners is found to differ widely, in some respects more widely than do the mortalities of any other occupational groups.

Considerable differences also exist in morbidity, especially with regard to certain special occupational diseases. Thus nystagmus, when related to conditions of work, is confined to coal-miners and has been found to exhibit a definite relation to dimness of illumination [1]; again subcutaneous cellulitis of the knee, elbow and hand, is seldom seen except among coal-miners. Knowledge with regard to the prevalence of these diseases depends upon what has come to light since they were included in the schedule to the Workmen's Compensation Act, 1906. On the other hand tuberculous silicosis is so prevalent among certain metalliferous-miners that knowledge of the disease depends to no small extent upon studies carried out in such occupations as tin-mining and gold-mining. To-day, however, owing to lack of reliable data the amount of invalidity occurring in the different forms of mining cannot be accurately determined. Hence for investigation into the health of the mining industry, reliance must be placed upon mortality records.

*Sources of Information.*—Information regarding mortality is given in the Decennial Supplements published by the Registrar-General. The first Supplement to provide sufficient detail was the one [2] dealing with the years 1890-92; then came the one [3] for 1900-01; and that [4] for 1910-12 has just been published. The information concerns (1) coal-miners, which group is further subdivided according to the main coal-fields—(a) Durham and Northumberland, (b) Lancashire, (c) Yorkshire, West Riding, (d) Derbyshire

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and Nottingham, which are given separately in the last report, (e) Staffordshire, which includes two coal-fields, that of North Staffordshire and that of South Staffordshire, and (f) Monmouthshire and South Wales; (2) ironstone-miners, from among whom hematite-miners are separated for 1910-12; (3) tin-miners; and (4) lead-miners. Copper-miners were dealt with in the two earlier reports, but the numbers at risk were too small to justify their further consideration.

TABLE I.  
*Mines under Coal Mines Act.*

Period	AVERAGE ANNUAL		Number employed per million tons raised	ACCIDENT DEATH-RATES		Period	Comparative mortality All causes
	Output (tons)	Number employed		per 1,000 employed	per million tons raised		
1873-1882	152,221,629	503,428	3,300	2.24	7.42	—	—
1883-1892	182,646,507	571,719	3,130	1.81	5.65	1890-92	1,068
1893-1902	215,790,835	732,391	3,390	1.39	4.70	1900-02	846
1903-1912	267,730,134	957,848	3,570	1.33	4.76	1910-12	727

Certain other information prepared by the Mines Department [5] is also available.

In coal-mining the population employed and the output have been rising since 1873 (*see* Table I); but a larger number is now required to give the same output; the accident mortality-rate since 1883-92 has fallen by 26.5 per cent. and the general death-rate by 31.9 per cent. The deduction seems fair that as the coal-miner has worked less strenuously, his tendency to die or be killed during the working period of life has diminished.

TABLE II.  
*Mines under Metalliferous Mines Act.*

Period	AVERAGE ANNUAL		Number employed per million tons raised	ACCIDENT DEATH-RATES	Period	COMPARATIVE MORTALITY ALL CAUSES		
	Output (tons)	Number employed				Iron miners	Lead miners	Tin miners
1873-1882	4,278,577	55,388	12,967	1.62	—	—	—	—
1883-1892	4,110,068	42,481	10,336	1.44	1890-92	893	1,514	1,628
1893-1902	3,637,745	33,669	9,255	1.31	1900-02	723	1,199	2,169
1903-1912	3,275,844	29,443	8,988	1.30	1910-12	652	1,185	1,579

In metalliferous-mining the population and the output have been diminishing (*see* Table II); a larger number are now needed to give the same output; the accident mortality-rate since 1883-92 has fallen by 9.7 per cent. (much less than for the coal-miner), while the general mortality which may be judged by falls of 18.6 per cent. for the iron-miner, 3 per cent. for the tin-miner, and 21.8 per cent. for the lead-miner, appears on the whole also to have fallen much less than that of the coal-miner. A tendency for the accident-rate and general mortality to move together is again apparent.

Elsewhere [6] attention has been directed to the way in which accidents vary with health; here, however, remark may be made that investigation of

the causation of fatal accidents among coal-miners does not, at first sight, point to health being the main factor concerned (see Table III). The trend shown in the figures indicates, says the Chief Inspector of Mines, "a very striking decrease in the number of accidents due to explosions and to shaft accidents, for the prevention of which much has been done by research and regulation, and an improvement less marked in the number of accidents due to falls of ground, in the prevention of which, though many are of course unavoidable, personal care plays a predominant part. On the other hand, no improvement is evident in the number of accidents on haulage, an operation in which safety depends far more on the personal factor than on regulation, and in which the work of the individual is not open to close supervision." In other words it is not enough for the natural protective mechanism against accidents to be in good order; the possessor of the mechanism must also be instructed how to act; the "Safety First" movement is required.

TABLE III.

*Death-rates from different causes of Accidents, per 1,000 persons employed underground in Mines under the Coal Mines Acts.*

Decennial period or year			DEATH-RATE CAUSED BY				
			Explosions of firedamp or coal dust	Falls of ground	Shaft accidents	Haulage accidents	Miscellaneous
Averages	1873-1882	...	0.65	1.12	0.32	0.32	0.16
	1883-1892	...	0.32	1.00	0.19	0.33	0.17
	1893-1902	...	0.18	0.76	0.13	0.29	0.16
	1903-1912	...	0.17	0.74	0.11	0.30	0.14
	1913-1920	...	0.51	0.68	0.11	0.28	0.16
1913	...	...	0.03	0.70	0.08	0.34	0.13
1914	...	...	0.05	0.89	0.08	0.35	0.17
1915	...	...	0.03	0.89	0.06	0.33	0.16
1916	...	...	0.02	0.89	0.08	0.38	0.12
1917	...	...	0.20	0.86	0.06	0.33	0.15
1918	...	...	0.03	0.62	0.05	0.25	0.11
1919	...	...	0.03	0.55	0.04	0.24	0.12
1920	...	...					

Before, however, the conclusion is accepted that improvement in general health has had but little influence upon the personal factor in relation to accidents, note must be taken of the large proportion of mining accidents now classified as "miscellaneous." In 1921 this miscellaneous group among coal-miners accounted for 29,360 out of a total of 78,475 underground accidents, and in 1920 for 35,360 out of 105,861. Investigation is showing that most of the accidents in this group are due (a) to men falling or hitting against stationary objects, or (b) to moving objects hitting men, or (c) to strains or sprains occurring while at work; all such incidents are affected by the personal factor. Only by close investigation of this "miscellaneous" group can the influence of the personal factor, as affected by health, upon the accident-rate among miners be determined. At present available information only refers to the miscellaneous group taken together; it indicates a tendency for this class of accident to have been rather less prevalent in recent years, but the fall rather accords with the small decrease in haulage accidents and is not of the same magnitude as the decrease in explosions and shaft accidents.

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TABLE IV.  
*Comparative Mortality from Certain Causes among Coal-miners, ages 25-64 inclusive.*

Coalfield	All causes	Phthisis	Pneumonia	Bronchitis	Diseases of Liver	Alcoholism	Accidents
1890-92							
Derbyshire and Nottinghamshire ...	841	80	77	87	21	3	104
Durham and Northumberland ...	894	109	85	58	26	5	111
Yorkshire ... ..	1,051	142	165	137	19	5	131
Staffordshire ... ..	1,100	95	127	204	9	2	157
Lancashire ... ..	1,236	118	217	198	20	6	179
Monmouthshire and South Wales ...	1,322	124	190	153	19	8	281
All Coalfields ... ..	1,068	113	141	131	19	5	163
Occupied Males (England and Wales)	1,102	214	122	101	32	15	64
1900-02							
Derbyshire and Nottinghamshire ...	675	64	52	49	22	2	80
Durham and Northumberland ...	763	84	54	41	17	6	105
Yorkshire ... ..	846	66	71	104	17	5	118
Staffordshire ... ..	783	88	71	67	12	5	99
Monmouthshire and South Wales ...	951	93	108	104	15	5	169
Lancashire ... ..	1,006	96	149	113	14	7	131
All Coalfields ... ..	885	89	86	79	16	5	123
Occupied Males (England and Wales)	925	175	87	53	25	16	58
1910-12							
Nottinghamshire ... ..	570	53	40	25	4	—	66
Derbyshire ... ..	591	70	34	39	7	1	73
Northumberland and Durham ...	635	70	54	33	8	1	83
Staffordshire ... ..	717	74	70	61	4	1	109
Yorkshire ... ..	758	81	69	45	8	1	117
Monmouthshire and South Wales ...	777	70	69	66	8	2	131
Lancashire ... ..	941	107	100	88	10	2	183
All Coalfields ... ..	727	76	64	51	7	2	118
Occupied and retired Males (England and Wales) ... ..	790	142	66	38	—	4	47

COAL-MINING.

Examination of the mortality from various causes among coal-miners, whether taken as a whole or when subdivided according to the separate coal-fields, during each of the three decennial periods under consideration, displays that some causes are of interest because the mortality ascribed to them much exceeds the standard, while others are of interest because their mortality falls distinctly below the standard. Causes not differing appreciably from the

standard (with the one exception of cancer) are disregarded in this paper as being of less interest. The causes with an excessive mortality are pneumonia, bronchitis and accidents; the causes with an unusually low mortality are phthisis and diseases attributed to alcoholism. When the separate fields are considered (Table IV) variations in the mortality from all causes are found, so great that in 1910-12 the comparative figure for Lancashire (941) is nearly double that (570) for Nottinghamshire. When death-rates from all causes are used for arranging the fields in order of merit, orders are produced which are nearly the same for each of the decennial periods. Further, if either of the selected causes of death (excluding the alcoholic group) is used instead of all causes the resulting orders are not much changed; and this applies particularly to the death-rates from accidents. The idea emerges that some common and persistent influence must be at work on some fields which results in a tendency to succumb to accidents, pneumonia, bronchitis and phthisis.

Geographical and climatological influences must first be considered, especially since the position of Lancashire, where the miners stand so uniformly high for the respiratory diseases, has been carefully considered by Greenwood and also by Stevenson [7], who have concluded that climate does exert an important influence in that part of the kingdom, causing an unduly high mortality from pneumonia and bronchitis. Reference to the general mortality of the districts also gives some support to the idea that climate is important. The death-rates from all causes and from phthisis, for males and females, are stated for the districts in Table V. The death-rates from all causes among males show a tendency to agree with the orders in Table IV; but the tendency is less pronounced when the death-rates from phthisis alone are considered.

TABLE V.  
*Mortality among the General Population in Coal-mining Counties (1901-10).*

County	Death-rate per 1,000 living					
	All causes		Phthisis			
	Males	Females	Males	Females	Males	Females
Derby ... ..	15.52	13.48	0.66	...	0.50	...
Nottingham ... ..	16.46	14.48	0.66	...	0.55	...
South Wales ... ..	18.18*	15.92*	0.66*	...	0.68*	...
Monmouth ... ..	16.77	15.11	0.53	...	0.54	...
Stafford ... ..	17.76	15.28	0.72	...	0.54	...
Northumberland ... ..	18.59	16.23	1.00	...	0.84	...
Yorks, West Riding ... ..	18.27	15.45	0.76	...	0.55	...
Durham ... ..	18.63	16.65	0.88	...	0.83	...
Lancashire ... ..	20.64	17.44	0.94	...	0.67	...
England and Wales ... ..	16.60	13.95	0.78	...	0.58	...

\* This relates to Glamorgan only where the bulk of the South Wales coalfield, outside Monmouth, lies.

Since the number of miners in some counties, such as Glamorgan, might be held to influence considerably the mortality among males, the mortality among females may, perhaps, be taken as a more useful measure of local influences, such as climate and density of population. The death-rates from all causes among females is found to be highest in Lancashire, and lowest in Derby and Nottingham; thus the position of these counties is in agreement with the order of Table IV. The magnitude of the differences between the highest and the lowest death-rates is far less than it is in Table IV, however, while the order of the other counties in no way conforms to that order. The death-

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rates from phthisis among females exhibit no similarity with the order of Table IV.

The conclusion seems fair that local conditions must be held to contribute something to the differences in mortality-rates found to exist among coal-miners, but that, particularly with regard to phthisis, inquiry must be made within the industry to ascertain why these differences are so accentuated.

*Phthisis.*—The question has recently been dealt with at some length [8] and the mortalities of different groups of miners—(a) those working at the coal face, (b) those employed otherwise underground, and (c) those working on the surface, on each coalfield have been ascertained and compared; the results need only be summarized here. The evidence was held to be compatible with the theory that the geological formation of the different coalfields was probably the common and persistent influence sought for. The silica content or some other mineral content of the coal worked and also of the roof of the workings would on this theory account for the variations in the mortality from phthisis and other respiratory diseases; while falls of ground, which form a high percentage of accidents, are related to the composition of the roof and the depth of the workings.

TABLE VI.

*Mortality from Phthisis per 1,000 living at various Age-periods in certain Occupations for Three Decennial Periods.*

Occupation	Periods	Age-period						
		15.	20.	25.	35.	45.	55.	65 and over
Printer ...	1890-92	1.42	4.21	4.97	<b>5.87</b>	5.22	5.30	4.39
	1900-02	1.03	3.45	3.65	<b>5.13</b>	4.34	3.68	1.87
	1910-12	0.78	2.60	2.64	3.07	<b>3.37</b>	2.90	2.19
Tailor ...	1890-92	1.20	2.81	3.44	<b>5.62</b>	5.26	3.34	1.88
	1900-02	0.00	2.11	2.64	4.12	<b>4.22</b>	3.43	1.79
	1910-12	0.54	1.81	2.24	2.83	2.86	<b>2.88</b>	1.78
Shoemaker ...	1890-92	1.08	2.73	4.00	4.32	<b>4.87</b>	3.14	4.55
	1900-02	0.03	2.95	3.27	<b>4.41</b>	<b>4.40</b>	3.21	2.15
	1910-12	1.20	2.20	2.96	3.21	3.49	2.83	1.88
Potter ...	1890-92	0.68	2.15	3.16	6.27	<b>7.51</b>	6.56	0.58
	1900-02	0.62	1.40	2.03	3.82	<b>7.26</b>	4.85	1.84
	1910-12	0.93	1.50	2.54	3.56	5.71	<b>6.03</b>	1.78
Cutler ...	1890-92	1.12	2.71	3.27	8.03	7.75	<b>8.08</b>	2.72
	1900-02	0.00	2.02	3.78	9.01	<b>11.09</b>	9.01	1.92
	1910-12	1.02	2.65	3.96	7.90	<b>9.21</b>	6.70	4.35
Tin- and lead-miner ...	1890-92	1.10	3.21	3.78	6.15	10.59	<b>13.85</b>	<b>13.89</b>
	1900-02	0.56	1.54	6.01	7.85	10.50	<b>12.22</b>	10.00
	1910-12	0.20	2.31	3.24	9.23	10.44	<b>13.72</b>	8.24
Sandstone mason ...	1910-12	0.00	1.03	2.36	6.43	8.92	<b>9.51</b>	0.75



TABLE VII.

*Mortality from Phthisis per 1,000 living at various Age-periods on different Coalfields for Three Decennial Periods.*

Coalfield	Period	AGE-PERIOD						
		15-	20-	25-	35-	45-	55-	65 and over
All coal-miners ...	1890-92	0.58	<b>1.39</b>	1.21	1.45	2.07	<b>2.23</b>	2.03
	1900-02	0.41	1.03	0.96	1.09	1.52	<b>2.04</b>	1.47
	1910-12	0.54	1.81	0.84	1.02	1.31	<b>1.43</b>	1.02
Derbyshire and Nottinghamshire ...	1890-92	0.40	0.84	0.98	1.00	1.24	<b>1.61</b>	1.26
	1900-02	0.32	1.13	0.64	0.77	<b>1.19</b>	0.92	<b>2.24</b>
	1910-12	0.59	0.80	0.84	0.59	1.05	<b>1.43</b>	0.46
Durham and Northumberland ...	1890-92	1.22	<b>1.98</b>	1.41	1.50	1.81	1.54	0.97
	1900-02	0.73	1.65	1.10	1.01	1.32	<b>1.70</b>	<b>1.71</b>
	1910-12	0.61	1.10	0.95	0.97	0.92	<b>1.14</b>	0.76
Yorkshire ...	1890-92	0.33	1.27	1.17	1.77	2.33	4.23	4.63
	1900-02	0.39	0.70	0.80	1.02	1.27	<b>1.87</b>	1.16
	1910-12	0.67	0.90	0.74	1.01	1.55	<b>1.84</b>	1.74
Staffordshire ...	1890-92	0.21	0.83	0.93	1.30	<b>1.95</b>	1.69	1.74
	1900-02	0.15	0.67	0.64	0.95		1.01	1.56
	1910-12	0.48	0.75	0.91	0.92	<b>1.36</b>	0.94	1.06
Monmouthshire and South Wales ...	1890-92	0.65	1.48	1.35	1.24	2.14	<b>2.94</b>	1.88
	1900-02	0.34	0.90	0.94	1.20	1.43	<b>2.34</b>	1.44
	1910-12	0.27	0.56	0.62	0.99	1.25	1.59	1.05
Lancashire ...	1890-92	0.35	0.96	0.96	1.62	<b>2.64</b>	2.17	1.79
	1900-02	0.35	0.89	0.81	1.25	<b>1.76</b>	<b>2.46</b>	2.19
	1910-12	0.45	0.67	0.94	1.56	<b>2.05</b>	2.01	1.27

Some evidence not previously discussed in favour of phthisis, when, as in Lancashire, it is comparatively prevalent among coal-miners, being related to the inhalation of dust, may be here dealt with. It is based on the fact that tuberculous silicosis occurs, not only in association with an undue prevalence of other respiratory diseases, but also at a later period of life than ordinary pulmonary tuberculosis [9]. A glance at the records (Table VI) for the "phthisis ridden" industries, tailoring, printing and shoemaking, discloses that the age of maximum incidence for phthisis in these trades is distinctly earlier than for men among whom silicosis occurs, e.g., lead and tin-miners, potters, sandstone masons, and cutlers. In each of these latter occupations the maximum incidence has for thirty years been late in life. The maximum incidence among coal-miners shows this same phenomenon (Table VII), a fact which of itself might raise the suspicion that the same influence may be at work. This suspicion is strengthened when the phthisis mortality on each of the coalfields taken separately is found to exhibit the same tendency. The type of phthisis appears to be the same and only to vary in intensity. Further support may also be found by considering the age of maximum incidence for phthisis among men employed on the surface, whose dust risk is much less; among this group of workers, 1910-12, the age of maximum incidence occurred for all coalfields and for Monmouthshire and South Wales at 35-44, for Derbyshire at 25-34, and for Durham and Northumberland, for Lancashire, for Yorkshire, and for Nottingham at 20-24. These last data do not support the hypothesis that the

late age of maximum incidence for phthisis among coal-miners is decided by local climatic or social conditions.

If the suggestion be accepted that phthisis, as it occurs among coal-miners, is similar in statistical type to the disease as found among workers exposed to silica dust, then it should be due to some similar influence; but some explanation is still needed to account for its unusually low prevalence among coal-miners. Possibly work carried out by Mavrogordato [11] may provide the needed clue; he exposed one group of guinea-pigs to silica dust alone, a second to silica dust *followed* by coal, and a third to silica dust *preceded* by coal. His results indicated that "once silica is fixed in the lung tissues, coal exerts no eliminative effect. If anything the influence is in an opposite direction, but a prior or even simultaneous exposure to coal dust appears to set up a condition in the lung which is inimical to the fixation of silica." No explanation is as yet forthcoming of how coal dust exerts this influence.

*Alcoholism.*—Interest also attaches to the group, alcoholism and diseases of the liver. Here the order of the fields, if arranged according to mortality from this group of diseases, bears no relation to that given by the other causes of death already considered. The reaction of the coal-miner to alcohol is curious; it was first pointed out by Sullivan [10]. The coal-miner is essentially a "convivial" drinker, as is shown by examining the records of convictions in 1911 for drunkenness per square mile in the counties of England and Wales. Middlesex, it is true, stands top, doubtless owing to its unique position with regard to the City of London; but it is followed by Durham (6'15); Glamorgan (6'13); Yorkshire, West Riding (3'62); Lancashire (3'32); Northumberland (2'37); Staffordshire (2'29); and Derbyshire (2'19). The existence of large coalfields is the characteristic common to each of these seven counties, which stand well above all the rest for convictions.

TABLE VIII.

*Comparative Mortality, 1910-12, for Alcoholic Diseases.*

Lancashire ... ..	12
Monmouth and South Wales ... ..	10
Yorkshire ... ..	9
Northumberland and Durham ... ..	9
Derbyshire ... ..	8
Nottingham ... ..	4

*Average Depth of Workings, 1922,\* in feet.*

Monmouth and South Wales ... ..	1,650
Lancashire ... ..	1,500
Yorkshire ... ..	1,220
Nottingham ... ..	1,220
Derbyshire ... ..	922
Durham and Northumberland ... ..	800

\* Staffordshire is omitted because the wide variations in the depth of the workings renders any average quite unreliable.

The miner is not an "industrial" drinker. No alcohol can be obtained after going below; when the effect of a dose taken before commencing work passes off, another dose cannot be obtained. The resulting effect is so unpleasant that the miner holds he cannot do his work on alcohol, and for the most part takes none until the day's work is over.

Herein he differs from such men as dock labourers, who throughout the day have recourse to refreshers. Mortality records indicate that in-

dustrial drinking, i.e., drinking before and during work, is far more harmful than convivial drinking. The coal-miner often works nearly stripped, in hot seams, and has to carry with him any fluid he consumes while at work; hence he frequently leaves the pit thirsty. Generally speaking, the temperature of the workings increases with their depth. The coalfields are arranged in Table VIII according to mortality from alcoholic diseases in 1910-12, and according to the average depth of the present workings. Since 1910-12 the mines in South Wales have been deepened below those in Lancashire, the transposition of these two fields is, therefore, not a matter of importance. As regards the rest, if the positions of Nottingham and of Northumberland with Durham were transposed, the orders arrived at would be identical. Probably the similarity shown by these two orders is not entirely a chance one, and the suggestion arises that the provision of drinking water underground might have considerable effect in lessening convivial drinking among coal-miners.

TABLE IX.  
*Proportionate Mortality from Cancer, 1910-12, according to Parts affected.*

Occupational group	Skin, penis and scrotum	Buccal cavity	Stomach and liver	Peri- toneum, intes- tines, rectum	Other parts not specified	All parts	Total number of cases
All males ... ..	5.2	7.9	32.2	19.7	35.0	100.0	46,198
Coal-miners ... ..	5.7	6.6	41.5	19.6	26.6	100.0	1,799
England and Wales—							
(i) above ground ... ..	7.1	9.5	36.3	19.7	27.4	100.0	168
(ii) at face ... ..	6.1	6.8	41.4	19.4	26.4	100.0	1,287
(iii) others underground ... ..	3.8	4.4	44.8	20.3	26.7	100.0	344
Northumberland and Durham—							
(ii) at face ... ..	5.6	4.2	44.4	17.6	28.2	100.0	284
(iii) others underground ... ..	4.5	0.9	49.1	16.4	29.1	100.0	110
(i) Lancashire ... ..	3.6	12.7	34.9	21.1	27.6	100.0	166
(i) Yorkshire ... ..	5.7	5.7	44.3	20.5	23.8	100.0	210
(ii) at face—							
Derby ... ..	9.9	4.4	39.6	26.3	19.8	100.0	91
Nottingham ... ..	7.4	9.3	33.3	27.8	22.2	100.0	54
Staffordshire ... ..	6.8	7.6	39.8	16.1	29.7	100.0	118
Monmouth ... ..	3.1	6.2	42.2	18.8	29.7	100.0	64
Glamorgan (South Wales)—							
(ii) at face ... ..	7.7	7.0	47.3	14.0	24.0	100.0	129
(iii) others underground ... ..	3.1	7.5	40.0	22.5	20.3	100.0	80
All other miners (tin, iron and lead)	2.7	3.8	51.0	17.5	25.0	100.0	184

*Cancer.*—The mortality from cancer when considered as a whole among different classes of miners does not present marked variations when compared with that for All Males; the only definite point seems to be that coal-miners suffer rather less than All Males.

In view of the high mortality experienced from cancer of the skin by chimney sweeps, by those who handle coal tar pitch, and by shale-oil workers, all of whom are exposed to the products of coal distillation, interest attaches to the prevalence of cancer of the skin among coal-miners. In order to investigate the point, information has kindly been provided by the General Register Office relating to the parts of the body affected. The data have been analysed at the National Institute for Medical Research, and are presented in the form of percentages of all cancer cases in Table IX.

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The coal-miner, according to this evidence, does not experience a high incidence of cancer of the skin; while the metalliferous-miners are definitely on the low side. The only proportion of all cases which is consistently, although not greatly, above the standard for All Males is that for stomach and liver: no explanation is hazarded to account for this point.

TABLE X.

*Comparative Mortality from certain Causes among Metalliferous-miners and others.*

Industry	Period	All causes	Phthisis	Pneumonia	Bronchitis	Other respiratory diseases	Diseases of circulatory system	Bright's disease	Diseases of liver	Alcoholism	Accidents
Tin-mining ...	1890-92	1628	586	123	166	146	109	34	33	4	56
	1900-02	2169	838	86	199	454	167	55	7	—	56
	1910-12	1579	684	95	76	144	126	54	—	—	95
Lead-mining ...	1890-92	1514	440	147	142	85	165	38	40	5	50
	1900-02	1199	317	84	56	131	117	13	21	6	68
	1910-12	1185	335	49	50	98	187	47	—	—	96
Ironstone-mining: — All districts	1890-92	893	104	120	87	29	97	17	23	4	100
	1900-02	723	126	67	41	31	90	8	19	6	117
	1910-12	652	73	76	37	23	98	21	3	2	93
Cumberland and Lancashire only	1910-12	983	123	114	80	66	153	25	—	6	104
Mason: Sandstone ...	1910-12	1427	415	120	116	75	172	58	14	4	77
„ Limestone ...	1910-12	753	129	54	38	18	130	33	8	5	54
Males, England and Wales: Occupied	1890-92	1102	214	122	101	32	145	31	32	15	64
	1900-02	925	175	87	53	25	135	32	25	16	58
Occupied and retired	1910-12	790	142	67	38	15	118	—	—	4	47
Boiler-maker ...	1890-92	1162	195	165	133	40	152	37	28	11	74
	1900-02	971	142	103	74	31	142	22	20	12	76
	1910-12	754	111	65	46	10	120	37	9	3	42
Slate-quarrier ...	1910-12	890	220	57	23	18	118	26	—	—	61

## METALLIFEROUS-MINING.

Mortality records for three different forms of metalliferous-mining are available: (i) Tin-mining, (ii) lead-mining, and (iii) ironstone-mining. Mortality records distinguish the first two from the last, not only on account of the excessive mortality from All Causes suffered by the tin and lead-miner at each decennial period, but because the excess in each case is due to the same causes, viz., phthisis with respiratory diseases, and, to a lesser extent, Bright's disease. The influence at work is known to be exposure to silica dust.

*Tin-miners.*—Veins of tin ore, which occur in this country exclusively in Cornwall, not only contain quartz, but lie in a rock largely made up of quartz: much dust is generated in working the rocks to obtain the ore. The mortality experienced is stated in Table X, in which the mortality from Bright's disease is also given. The high rates from phthisis and other respiratory diseases are in accord with what is now recognized to occur among men exposed to silica

dust. The late age-incidence of this form of phthisis has already been pointed out. Recent work by Gye, Purdy and Kettle [12 and 13] on the toxicity of colloidal silica and its influence in determining fibrosis and tuberculosis lends interest to the somewhat high rates experienced for Bright's disease. The phenomenon is present in the death-rates for sandstone masons but not for limestone masons. The conclusion suggested is that silica dust becomes gradually converted into soluble silica in the lungs; it causes the well-recognized fibrotic changes in the pulmonary tissues; but some escapes into the blood to be excreted by the kidneys which are similarly affected during the process.

TABLE XI.

Deaths which occurred among Lead-miners in Wales, 1908-18.\*

Age group	Cause of death	Number of deaths		Value of P.
		Expected	Observed	
25-35	Phthisis	2.3	3.5	P = 0.54
	Other respiratory diseases	0.6	0.0	
	All other causes	5.1	4.5	
	All causes	8.0	8.0	
35-45	Phthisis	3.2	4.0	P = 0.79
	Other respiratory diseases	0.8	1.0	
	All other causes	3.5	2.5	
	All causes	7.5	7.5	
45-55	Phthisis	8.2	14.0	P = 0.04
	Other respiratory diseases	4.1	2.0	
	All other causes	9.2	5.5	
	All causes	21.5	21.5	
55-65	Phthisis	8.4	23.5	P = 0.0000
	Other respiratory diseases	7.7	5.5	
	All other causes	20.4	7.5	
	All causes	36.5	36.5	
65 and over	Phthisis	0.7	14.5	P = 0.0000
	Other respiratory diseases	9.7	7.5	
	All other causes	25.6	14.0	
	All causes	36.0	36.0	

\* Where the age at death was on the line between two periods, e.g., at 25, 35 or 45, half a death has been credited to each period.

*Lead-miners.*—Lead ore is found in different districts; the mineral occurs as galena (sulphide of lead) together with quartz crystals, in thin veins. These veins may occur in a granite country rock, as at Greenside, Patterdale, or in a slaty country rock, as near Keswick, or in a limestone rock. The phthisis mortality varies: at Greenside the men suffer excessively; at the mine near Keswick no excess was found. Some idea of the excess which may occur may be estimated from Table XII (prepared by Dr. Greenwood from data supplied to him) dealing with deaths which occurred among lead-miners in Wales. The mortality from Bright's disease among lead-miners is also rather above the standard. When these records are being considered the paucity of the data owing to the small populations at risk must be kept in mind; hence the low figure for Bright's disease for 1900-02, which is

based on only seven deaths, may be fallacious. In neither of these mining groups is there any sign of an excessive mortality from alcoholic diseases which, were it present, might be the agent concerned in the high mortality from Bright's disease.

TABLE XII.

*Mortality from Phthisis per 1,000 living at various Age-periods in Ironstone-mining for Three Decennial Periods.*

Occupation	Period	Age-period						
		15-	20-	25-	35-	45-	55-	65 & over
All ironstone-miners ...	1890-92	1.49	0.78	1.81	<b>1.71</b>	1.67	1.08	<b>2.05</b>
	1900-02	0.82	0.57	1.22	1.43	2.08	<b>3.65</b>	2.46
	1910-12	0.29	0.74	0.65	<b>1.39</b>	1.03	<b>1.25</b>	0.61
Cumberland and Lancashire ...	1910-12	0.0	0.63	0.81	<b>2.72</b>	1.51	<b>2.55</b>	1.51
	1910-12	0.43	0.79	0.59	<b>0.90</b>	0.86	0.49	0.00
Boiler-makers ...	1890-92	0.49	1.49	2.30	2.39	3.24	<b>3.92</b>	1.81
	1900-02	0.31	0.88	1.68	2.45	2.17	<b>2.51</b>	1.45
	1910-12	0.33	0.78	1.19	1.75	1.40	<b>2.45</b>	1.69
Slate-quarriers ...	1910-12	1.16	2.43	2.11	2.05	2.83	<b>7.91</b>	7.05

*Ironstone-miners.*—The group, ironstone-miner, is of interest. In the first period the maximum incidence of phthisis appears at 65 and over, but there was an earlier rise at 35. In the second period the disease had risen in prevalence; it shows a high incidence at 65 and over, and a maximum at 55. So far the group agrees fairly with the other mining groups, and would support the idea that mining *per se*, apart from any dust hazard, is associated with a late age-incidence for phthisis. But the third period differs from the other two; the disease has fallen strikingly in prevalence yet the maximum incidence is at 35, with a secondary rise at 55. Information concerning this third period throws light on the position. There are two kinds of iron ore: one hematite, or oxide of iron, found associated with limestone rocks; the other ironstone or carbonate of iron, found associated with oolitic limestone. Hematite only is obtained in Cumberland and North Lancashire; and the miners in this district, therefore, represent exposure in getting this type of ore. Data are given separately for this group for the third period. When these data are subtracted from the whole group, no sign is left in the remainder of any late-age high incidence of phthisis; the maximum is at 35, falling thereafter with age (Table XII). This remainder is fairly representative of men employed in getting jurassic ironstone or carbonate of iron whether at Cleveland or other places. Hence mining *per se* is not the determining influence of the late age-incidence noted. The hematite group, it is true, also shows a maximum incidence at 35, but it also shows a marked second rise at 55. In the earlier periods this second rise may have been more pronounced. In those periods the hematite group formed a larger proportion of the whole group of iron-miners, comprising 42.3 per cent. in 1891, 36.6 per cent. in 1901, and only 29.8 per cent. in 1911.

Concentrating attention on the records for 1910-12 the Cumberland and Lancashire miner has a higher rate than the rest of the ironstone group for

phthisis together with other respiratory diseases and for Bright's disease (Table XIII); while the maximum incidence for phthisis is at a later period. Here are displayed, in modified form, statistical characteristics first recognized in relation to workers exposed to silica dust. No clinical or pathological evidence can be quoted in support of tuberculous fibrosis occurring among these men; X-ray photography, it is true, has disclosed the presence of shadows suggestive of fibrosis in the lungs of these hematite-miners, but the opacity of oxide of iron to X-rays rather detracts from the significance of these findings. Nevertheless the suggestion does emerge that possibly the dust of oxide of iron may exert in a modified way a similar influence to that of silica.

TABLE XIII.

*Comparative Mortality, 1910-12, from Certain Causes among Ironstone-miners.*

Group	All causes	Phthisis	Pneumonia	Bronchitis	Bright's disease
All ironstone-miners	652	73	76	37	21
Cumberland and Lancashire	982	123	114	80	25
All other ironstone-miners	460*	53*	61*	16*	20*

\* These figures have been calculated, and are not given in the Registrar-General's tables.

Anyone who has visited these Cumberland hematite pits and has, thereafter, blown his nose, can have no doubt that exposure to dust of oxide of iron exists; the mucus expelled is entirely rusty in colour. Attention may also be directed to the staining of the pulmonary tissue which results, and has been referred to elsewhere [9].

#### OTHER OCCUPATIONS WITH EXPOSURE TO DUST OF OXIDE OF IRON.

The suggestion that dust of oxide of iron may have some influence receives support from consideration of the non-mining industrial group of boiler makers. (Table XII.) Mortality records for this group exhibit a maximum incidence for phthisis as late as 55-64 for each decennial period. The total death-rate from phthisis is not high, and in this respect the boiler maker resembles the hematite miner. Reference to mortality from respiratory diseases and from Bright's disease discloses that for 1890-92 the rates for boiler makers for pneumonia, bronchitis and other respiratory diseases, and for Bright's disease, were above the standard; for 1900-02 the same holds good, except that Bright's disease was not in excess; in 1910-12 bronchitis and Bright's disease were in excess with pneumonia equal to the standard. At each period the alcoholic diseases were below the standard. There is then evidence for boiler makers that the type of phthisis resembles that for hematite miners, and boiler makers are certainly exposed to dust of oxide of iron.

Other workers exposed to dust of oxide of iron are Sheffield cutlers (as distinct from grinders); and metal dressers and glazers. In neither case, however, is oxide of iron the only constituent of the dust. Complete mortality records for these occupations are not available; but a group of cutlers was found with a distinct excess of phthisis having a median age at death from 45-46 (as compared with from 38-39 for occupied and retired males); with a decided excess of bronchitis, and a slight excess of pneumonia. The position with regard to metal dressers and glazers is somewhat the same as that for the Sheffield cutlers [9].



The evidence obtained by reference to mortality records from these other industries is not against the inference suggested by the statistics of hematite miners that oxide of iron may not be an entirely harmless dust. In this connexion interest attaches to the fact that oxide of iron resembles silica in forming a positive colloid, in which form it is even more active than colloidal silica when brought into contact with living material.

#### ALUMINA.

Should a clue exist here, then other materials which form positive colloids might be expected to have a somewhat similar action. Alumina is known to possess this property; slate is compressed alumina, and slate quarrying provides an occupation with exposure to dust of this material. This occupation was not separated from that of stone quarrying in the two earlier Decennial Supplements, and any indications given by the statistics might be due to silica dust generated in quarrying sandstone.

Fortunately, however, the occupation of slate quarrying has been dealt with separately in the last Supplement. (Table XII, p. 96.) The total number of workers is not large and the mortality found must be accepted with caution. The comparative mortality for phthisis (220) among slate quarriers, although much lower than that (415) for sandstone masons, is much above that (129) for limestone masons; and the disease exhibits a clear tendency to have a maximum incidence late in life. No high mortality is found due to other respiratory diseases, or to Bright's disease; nevertheless the evidence is by no means adverse to the explanation that, so far as the tubercle bacillus is concerned, dust of alumina may possess a similar influence to that exerted by silica.

In this connexion Dr. J. S. Haldane permits me to say he has found through experimental work, as yet unpublished, in which animals were exposed to dust of alumina in the form of pure china clay, that cytological changes result similar to those obtained by using silica dust.

#### GANISTER MINING AND BRICK MAKING.

A point already alluded to in connexion with the possible influence of coal dust in lowering the incidence of phthisis among coal-miners, receives some support from the ganister industry. Ganister is a quartzite rock found underlying coal and overlying fireclay. Best ganister, which contains over 95 per cent. of quartzite, is made into first grade refractory bricks and other material used for lining steel furnaces. The dust to which those employed are exposed is nearly pure silica and they experience an excessive mortality from phthisis. Impure ganister which contains veins of fireclay is made into second grade refractory materials used for coke ovens; the dust contains about 80 per cent. of pure silica, but yet those employed do not experience an excessive mortality from phthisis. The addition of fireclay appears to interfere with the usual influence of silica.

The theory has been put forward [14] that the influence of coal may be due to some organic substance, and that this substance may be washed down from the coal into the underlying fireclay where it has been retained. Hence the clay comes to contain this hypothetical substance in concentrated form.

Fireclay, when freshly mined, is not fully plastic, i.e., not fully colloidal, but it becomes so after being exposed to weather for some years. During this weathering process some change occurs, possibly the removal by rain of some soluble substance. Hence it has been argued that freshly mined fireclay may contain some such water-soluble substance as humic acid, which interferes with

its tendency to become colloidal. Silica is held to exert its harmful influence in the human body by first becoming colloidal. A substance which interferes with the colloidal nature of clay might possibly, when inhaled with silica dust, also prevent silica from becoming colloidal when it would tend to be eliminated from the lungs as are other inorganic dusts. This idea may be entirely wrong, but, even if so, the facts it is called in to explain still remain.

*Low Infectivity.*—One further point with regard to tuberculous silicosis was first noted in relation to the disease as observed among metalliferous miners. The disease appears to exhibit a low power of infecting contacts not exposed to silica dust, such as the wives and families of the affected miners. Evidence bearing on this matter has been stated elsewhere [9]; it has recently been added to by Hoffman's investigations among granite cutters in America [15]. The meaning of this low infectivity has not yet been investigated; yet it presents an interesting problem.

#### SUMMARY.

(1) Similarities in occupational environment make the mining industry an interesting field for investigating mortality.

(2) Among coal-miners (a) the incidence of phthisis, even though low, and of other respiratory diseases, which is often high, is probably influenced by exposure to dust; (b) the habit of convivial drinking may be connected with lack of facilities for obtaining drinking water while working under conditions provocative of loss of moisture; and (c) the total cancer rate, including that for cancer of the skin, is low; but that for cancer affecting the liver and stomach is somewhat above the standard.

(3) Tin-miners and lead-miners suffer from typical tuberculous silicosis, with which is associated high mortalities from other respiratory diseases, and Bright's disease.

(4) Phthisis among hematite miners appears statistically to resemble in a modified way fibroid phthisis, which may be due to oxide of iron. Other workers, such as boiler makers, exposed to oxide of iron, also exhibit a similar statistical type of mortality.

(5) Carbonate of iron miners do not exhibit the same type of mortality.

(6) Alumina dust, judging from mortality data for slate quarriers, resembles oxide of iron and silica in its effect on the lungs.

(7) Ganister workers present an excessive phthisis mortality similar to that of tin-miners, unless fireclay is mixed with the ganister.

(8) Tuberculous silicosis exhibits a lower power of infecting contacts not exposed to silica dust.

#### DISCUSSION.

Dr. R. J. REECE (President) said that it had been generally known that the death-rate among coal-miners was not a heavy one, but Dr. Collis had put clearly before them that there was a wide difference in the mortality statistics of different classes of miners. The information collected in his paper afforded ample opportunity for study. His first table (p. 86) in which he showed that a larger number of miners were now required to produce the same output of coal than had formerly been the case was interesting, and with this was associated a general improvement in the health of the miner. His paper showed that the highest death-rate from all causes occurred in the coalfields that had the deepest mines, i.e., in South Wales and Monmouthshire, and in Lancashire. The Lancashire coalfields had also the highest death-rates for pneumonia and bronchitis over a series of years, and the South Wales and Monmouthshire coalfields closely followed those of Lancashire in this respect. It was a question whether the higher

temperature of the deeper mines was sufficient to account for this higher death-rate and whether the ventilation of the deeper mines played some part in its causation. From Table IV (p. 88), given by Dr. Collis, it would be seen that while the phthisis death-rate, in the period under review, showed little variation in the Lancashire coalfields, there was an appreciable fall in some of the others.

The late Dr. R. J. Ewart (whose recent sudden death they all deplored) in a paper which he read before the Section last year had called attention to the rise in the consumption of food and increase in wages in the country being closely allied to a corresponding fall in the death-rate from phthisis, and Dr. Collis, in his Presidential Address to the Tuberculosis Group of Medical Officers of Health, had pointed out that the consumption of milk and butter from 1860 to 1913 increased in a way that was comparable with the fall in tuberculosis. It was well known that the coal-miner had never stinted himself in the matter of food, and it would be of interest to know whether there was more food consumed in the coalfields than in other parts of the country, and whether the consumption of milk and butter had varied during the last thirty years in the Lancashire coalfields.

Could Dr. Collis tell them whether there was any material difference in the mortality of miners in different parts of the same coalfield? He (the President) had in mind the different character of the coal in the South Wales coalfield. Did those miners working in the anthracite coalfield, where presumably there was less dust, but possibly dust of a more irritating character than ordinary coal dust, suffer more from pulmonary complaints than those who worked in mines where coal was less hard? The work of the miner was very strenuous and it would be interesting to know whether the favourable life statistics of the coal-miner were in any way due to those who, in early life, felt their vigour abating and changed their occupation to work requiring less physical effort, such as tailoring or shoe-making. As regarded the number of convictions for drunkenness in different parts of England and Wales, it would be useful to know whether the efficiency or common-sense of the local police had been taken into consideration.

Dr. A. K. CHALMERS asked whether Professor Collis had had an opportunity of correlating his death-rates with the differing depths of shaft and methods of ventilation adopted in the mines in different parts of the country. With regard to the low infectivity-rate among contacts to which Professor Collis had referred, he thought a similar illustration occurred in Aberdeen, where, according to Dr. Hay's inquiry, the granite worker, although himself subject to a special incidence of phthisis, did not communicate it to anything like a corresponding extent to his wife or family.

Dr. DAVID MCKAIL suggested that the inference from the accidents due (a) to men falling or hitting against stationary objects, or (b) to moving objects hitting men, was that the better and more efficient lighting of mines would obviate most of these and bring the fall in these groups into line with the decrease in those due to explosions and shaft accidents.

Dr. MAJOR GREENWOOD: I have hardly anything to say of a critical nature, since almost all the little I know of these matters has been learned from my friend, Professor Collis. A comparison of regional rates of mortality from respiratory diseases shows that all areas in the north and north-west tend to suffer more severely than the midland and western areas, so that climate is probably an important factor. But a comparison between Cumberland and Lancashire establishes a probability that climate is not the only, nor perhaps even the most, important factor. The *relatively* higher mortality from cancer of the liver and stomach of the coal-miners is a point of interest; it would be valuable to ascertain whether a similar excess characterizes other occupations wherein convivial rather than industrial drinking plays a part.

I should like to supplement the words used by the President and to express my sense of the loss epidemiological science has suffered through the death of Dr. R. J. Ewart. Dr. Ewart was a distinguished member of the tiny band of medical statisticians. His work was characterized by a courage in facing difficulties and an originality of thought which are qualities we can ill afford to lose. To his personal friends, one of whom I had the honour to be, the loss is especially severe.

Dr. COLLIS (in reply) said that, while the data for some coalfields with regard to depth of workings and mortality increased together, yet on the Nottingham and Yorkshire fields where the depths were the same the comparative mortalities differed greatly, varying from 570 to 758 in 1910-12: so that, while depth, which might be considered some measure of difficulties in ventilation, might exert some influence upon health, it probably was not a major one. He associated himself with the President and Dr. Greenwood in mourning the loss of a keen and enthusiastic colleague and a generous scientist in Dr. Ewart, of whose untimely death he had only just learnt.

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*Addendum.*—A recent investigation [16] by Dr. E. L. Middleton (published while this paper was in the press) into the effect of dust-inhalation upon cutlers, dressers and glazers in no way runs counter to the suggestions advanced above.—E. L. C.



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### The Medical Services of Henry the Fifth's Campaign of the Somme in 1415.

By G. E. GASK, C.M.G., D.S.O., F.R.C.S.

THERE is not a great deal known about the medical arrangements made by Henry V for the care of his troops during his campaign in France in 1415, but, at least, some of the names of the medical men who served with him have come down to us, and it may be of interest to put together the few facts which are known. To those who participated in the Battle of the Somme in the late war, the accounts of Henry the Fifth's famous march from Harfleur round the head waters of the Somme to Agincourt, appear strangely fresh, and it is possible to visualize and sympathize with his hungry, sick and weary force, and to rejoice over his foolhardy but glorious feat. To understand the campaign, it is necessary to relate briefly the circumstances which led up to it.

Henry V began to reign on Tuesday, March 21, 1413. He was then 25 years of age and described as tall and slender with well-formed limbs. He was an athlete, a good soldier, trained in the hard school of the Welsh wars and in general had the qualities which made for popularity in a prince. Whatever his habits as a young man—and there are stories of his early wildness which do not appear to be substantiated—as king he worked hard and showed sterling qualities which earned for him the appreciation of his subjects.

Henry seems to have had two objectives in his mind, the first being to conquer France and the second to free Jerusalem from the infidel. The justice of Henry's claim to the crown of France is not the concern of this paper, but it seems to be a fact that he genuinely believed that his claim was a good one and was determined to fight for it. At Michaelmas, 1414, a Council of the magnates of the kingdom was held at Westminster when Henry asked their advice concerning his claim to the crown of France. They recommended that he should send ambassadors to France to negotiate, and in the meantime, that every preparation should be made for invading that kingdom.

It is clear that preparations were made for war on a large scale, for on September 22, 1414, Nicholas Merbury, as Master of the Ordnance, had been commanded to secure stone-cutters, sawyers, and all necessary workmen for the manufacture of guns and engines. Four days later, orders were issued forbidding any trader to export gunpowder from any English port, and by the

end of October 10,000 gunstones had been forwarded to London. It was essential for the safeguarding of the expeditionary force that Henry should have command of the sea, and so on January 30, 1415, orders were given for the King's ships to be manned, and it was arranged that the sea should be guarded from Plymouth to the Isle of Wight and from Orford Ness to Berwick during the king's approaching voyage, so that he might leave the country with an easy mind.

The first embassy to France came to nought and a second was sent, with no better results. War became imminent and preparations were pushed on. The feudal levy was not suitable for a prolonged French war, so Henry left to them the defence of England and raised an expeditionary force consisting of volunteers under contract or "indenture." By these indentures the King contracted with various nobles and knights to give their services and to provide, as well, certain numbers of men-at-arms and archers. In this way 2,500 men-at-arms and 8,000 archers were raised, and in addition there were numbers of non-combatants such as carpenters, smiths, miners and gunners, armourers, yeomen of the pavilions, bowyers, saddlers, physicians, surgeons and chaplains.

The chief surgeon to the force was Thomas Morstede, of London, and the "indenture" made with him is preserved. It commences thus:—

"Indentura cum Chirurgico Regis & Retinentia sua.

*Ceste Endenture*

Fait parentre le Roy nostre Souverain Seigneur d'une part, & Thomas MORSTEDE, Surgien de mesme nostre Seigneur le Roy, d'autre Part, Tesmoigne, que ycellui Thomas est Demorez devers nostre dit Seigneur le Roy, pour Lui servir, par un an entier, en un Viage, que même nostre Seigneur le Roy ferra, en sa propre Person, si Dieu plest, en son Duchee de Guyenne, ou en son Roiaume de France."

In what follows we read that Thomas Morstede was directed to be ready to attend a muster in the month of May (1415). He was to bring with him fifteen persons, of whom three should be archers and the others men of his own craft (mestier). In case the said Thomas should go into the Duchy of Guienne, he should draw for himself 40 "mares" and as wages for each of the said fifteen persons 20 marcs for the whole year. And if in the company of the King the said Thomas should go into the Kingdom of France, he should have for himself 12 deniers and for each of the said fifteen persons 6 deniers per day for the said year.

The wages were to be paid in advance for the first quarter when the muster had been made. As surety for the wages of the second quarter the King agreed to hand over jewels to the value of the amount of wages due—which jewels could be redeemed by the King later or if unredeemed could be sold by Thomas without hindrance from the King or his heirs after a year and a half and a month. Morstede was to be ready for a voyage over-seas with his retinue well mounted, armed and arrayed by the first day of July (1415). The King undertook at his own cost the trans-shipment of Thomas, his retinue, horses, harness and victuals. It was specified that in the event of the King of France or any of his relatives being taken prisoner by Thomas or any of his retinue, the prisoner should belong to the King—and for any other prizes of war, the King should take a third.

"Don. A Westm. le XXIX Jour d'Averill, l'an du regne du Roy nostre dit Seigneur Tierz."

On May 26, 1415, appeared a document from Thomas Morstede applying for money to purchase medical stores (unspecified) as well as one cart and two horses (somers) to carry the said stores. The request was granted. In another petition, undated, Morstede prays the king "to grant his letters of Privy Seal directed to your Chancellor of England to cause him to deliver to your suppliant letters of commission under your great seal by force of which he should have power to press, as well within as without franchise, twelve persons of his craft such as he should choose to accompany him and serve your most sovereign lord during your campaign."

To our modern ideas one cart and two horses seem ludicrously inadequate to carry all the medical stores for an army preparing for a year's campaign. It is well to remember, however, that in 1808, at the beginning of the Peninsular War, only a little over one hundred years ago, when Sir Arthur Wellesley landed in Mondego Bay, it appears in General Orders that the head of the Medical Department obtained two carts drawn by bullocks for the conveyance of the stores necessary for the army, which he loaded with a certain quantity of bearers, tin cups, spitting pots and other pots, none of which did or could arrive either at the place or at the time that they could be wanted; and nothing could be more inefficient than the medical department of the army during the first two-thirds of that war.

The physician to Henry V was named Nichol Colnet and an indenture was made with him on April 29, 1415. This indenture corresponds almost exactly with that made with Thomas Morstede the surgeon, and his wages were the same, but he was named alone and was to be accompanied by three archers and no other physicians.

The reason why both the physicians and surgeons were attended by archers is not quite clear. It might be that it was to swell the army, but it seems more probable that they were meant as a protection—for at that time there was no Geneva Convention. These indentures are not peculiar to the members of the medical corps, but they are found referring to other men, and the article referring to the possible capture of the rival sovereigns seems to be a usual formula.

Thus far we have records of the appointment of one surgeon and one physician, but in the History of the Battle of Agincourt, by Sir H. Nicolas, K.H. (1832), there appears in the list of the retinue of the King, present at that battle, the names of Thomas Morstede and William Bradwardyn (surgeons), each with nine more surgeons. This completes the list of medical officers as far as we know it, though it is possible and probable that some of the dukes and nobles may have taken with them their private medical attendants.

All preparations having been completed, the expeditionary force set sail from Southampton at three o'clock in the afternoon of Sunday, August 11, 1415, and landed on Wednesday, August 14, without opposition, close to Harfleur, at the mouth of the Seine. The Siege of Harfleur was begun on August 17, 1415, and the town was surrendered on September 22, after a considerable amount of hard fighting. The number of killed and wounded among the English seems to have been few, though no figures are given. At any rate there is no mention of the death from wounds of any man of note.

On the other hand the deaths and casualties from sickness were very high. It is stated that 2,000 died from the flux, one-fourth of whom were knights and squires, including Bishop Courtnay and the Earl of Suffolk, and 5,000 more had to be invalided home, among whom were the Earl Marshal and the

Earls of March and Arundel. The sickness which wrought such havoc with the troops is described as the flux, the bloody flux, or "*cours de ventre*." It is pretty clear it was a form of acute inflammation of the bowels accompanied by severe diarrhoea and passage of blood.

It is evident that the disease was epidemic, and affected both the officer or knightly class as well as the common soldier. The disease ran an acute course, being often fatal in a few days. These facts render it almost certain that the disease was what is now called dysentery. There is no evidence to show whether the disease was due to the amœba of dysentery, which caused such trouble to our troops in Mesopotamia during the recent war, or to the bacillary form. At any rate, it seems to have been a usual one in the country, for at the siege of Arras in 1414, then defended by the Duke of Burgundy, it is stated that 11,000 men died of the flux. During the late war it must be remembered also that in the Somme area both the British and the Germans suffered severely from dysentery of a rather mild and non-fatal type.

The conditions under which the English fought under Henry V were most favourable to the outbreak of bowel complaints, the great curse of all armies. It is chronicled that the English were short of food, and that the victuals they had brought with them were spoilt by the air of the sea. The men drank inordinately after working in the sweltering heat. Failing their usual rations, they ate too freely of unripe grapes and other abundant fruit. On fish days they greedily devoured the cockles and mussels that swarmed that year in the muddy creeks, and when the chilly nights succeeded to the hot autumn days, they lay down where the offal of slaughtered beasts lay rotting in the surrounding swamps. Hence fever, flux and dysentery struck down high and low alike. Nothing is said of flies, but we can imagine only too well how they swarmed over from putrefying carcases to the food and infected all. At the end of the siege of Harfleur it is estimated that over 2,000 died, of whom one-fourth were knights and squires, and 5,000 more had to be invalided home, and so Henry's army was sadly reduced in numbers.

There is no mention in the chronicles consulted of any treatment given or sanitary measures taken to combat the sickness, nor is it considered likely that there were any camp hospitals. The first mention of field hospitals occurs in the War of Grenada (1483-87) by the Spanish against the Moors.

Prescott in his "*History of the Reign of Ferdinand and Isabella*" states that:—

"Isabella, solicitous for everything that concerned the welfare of her people, sometimes visited the camp in person, encouraging the soldiers to endure the hardships of war, and relieving their necessities by liberal donations of clothes, and money. She caused also a large number of tents, known as 'the Queen's hospitals,' to be always reserved for the sick and wounded, and furnished them with the requisite attendants and medicines at her own charge. This is considered the earliest attempt at the formation of a regular camp hospital on record."

Harfleur surrendered after a vigorous siege, lasting about thirty-six days, and on September 23, Henry entered the town. According to the chroniclers, St. Remy and Monstrelet, Henry dismounted at the gate, took off his shoes and stockings and proceeded barefoot to the church of St. Martin, where he gave solemn thanks to God for his success.

On or about October 4 Henry held a Council of War to decide on his next move. Henry was advised to re-embark his troops and return to England. To this reasonable advice the King turned a deaf ear. He replied that he was

anxious to view the territories which were by right his own; that his trust was in God; that if he quitted Harfleur in the manner proposed the enemy would reproach him with cowardice. Calais was his objective and to Calais he would go.

On October 6 or 7 he set out on his adventurous march with his small army numbering about 6,000 men, or, on the most generous computation, 9,000 men. The troops carried with them food for eight days only. All baggage wagons were left behind and such stores as they took were carried on the back of sumpter horses. In a similar way the King's crown, state jewels and chancery seals were packed. The route taken was via Montivilliers and Fécamp and so along the coast route to the Somme, which they meant to cross at Blanchetache as Henry's ancestor Edward III did before the battle of Crecy. Montivilliers was left a little to one side; at that place there was some little opposition, for Nicholas states that a lancer named Geoffrey Blake was killed there. On October 11 the army arrived at Arques near Dieppe, where a supply of bread and wine for the troops was forthcoming under a threat of burning.

On October 12 a similar threat brought refreshments to the troops from the inhabitants of the town of Eu. On the next day, October 13, when the army expected to cross the Somme at Blanchetache, information was received that the ford was strongly held by the enemy and by a council of war it was decided to march higher up to Abbeville, but on arriving there to their great disappointment they found the bridges broken down and the French collected to prevent their passage. There was nothing to be done but to try higher up still and perhaps march to the head of the river which was estimated to be 60 miles distant. The river Somme runs a tortuous course through a broad valley; it is still surrounded with deep lagoons and swamps and marshy meadows overgrown with reeds and poplar trees, in which ducks and heron find good quarters. Before the river was canalized the swamps and lagoons must have been much greater and the river impassable unless bridged, or at recognized fords. It is clear that there was a ford at Blanchetache and bridges at Abbeville and Pont Rémy and almost certainly at Amiens and Corbie.

Apparently therefore Henry and his army took the road along the left bank of the Somme and examined the crossing at Pont Rémy, but there also the bridges and causeways were broken and the French showed themselves in force on the other side. They halted at Hangest and Crouy and the chroniclers recount their baffled and despondent mood. It is easy for us to-day to imagine the very field in which the men cast themselves down in weariness of body and despair of mind, for was it not there that troops detrained during the Somme campaign of 1918, and in the fields of Crouy, at the same time, that we had two casualty clearing stations.

The army must have marched next through Picquigny, the scene of the meeting just sixty years later, of Edward IV of England and Louis XI of France. Skirting Amiens on October 16 the army reached Boves, where the castle belonged to a partisan of the Duke of Burgundy. There they got food and drink: the archers exceeded their allowance and a scene of riot followed so that the King gave orders to stop the drink. "What need?" said someone to him: "the brave fellows are only filling their bottles!" "Their bottles!" replied the King in disgust, "they are making big bottles of their bellies and getting very drunk!"

The ruins of the castle of Boves remain to this day and the small town at the foot of the castle is occupied, according to Joanne's Guide, by "*presque tous*



*blanchisseurs.*" During the battle of 1916 it was here that the Fourth British Army got its washing done.

From Boves Henry must have pushed on up the old Roman road, which runs in almost a straight line to Vermand; he probably followed this as far as Villers Bretonneux, famed for its defence against the great German advance in March, 1918. From here he must have branched off to the left along a road which to-day runs down through pleasant wheat fields, to appear on October 17 outside the walled town of Corbie, a town well known to the British Expeditionary Force in 1916 as a billeting area and the site of two casualty clearing stations. Here the little river Ancre joins the Somme. A bridge must have been there for hundreds of years; and here, presumably, Henry thought that he might be able to cross, but a spirited sally by the French drove him off. Quitting Corbie Henry left the Somme, which there takes a very tortuous course, and avoiding the Chipilly bend he made for Nesle (or as the modern English soldier calls it—"Nestle"). On the way one of his soldiers, named Bardolph, according to Shakespeare, was discovered to have stolen a copper-gilt pix from a church which is believed to have been at Harbonnières.

At Nesle Henry received information that there were two places at which the river was capable of being passed; the approach to these was by two long but narrow causeways which, though broken in the middle, had not been destroyed by the men of St. Quentin, as ordered by the King of France. The places mentioned are Bethencourt and Voyennes. The crossing is described with minuteness. Nothing could surpass the personal exertions of the King: he caused the broken fords of the causeway to be repaired and stationing himself at one entrance and some officers on the other to preserve order and prevent crowding, he hurried his army across, so that though the head of the column only started at one o'clock they had all crossed by an hour before night. The passage was disputed by a few horsemen who retired before the advance guard and the army passed a joyful night in the very farm houses that had been occupied by the enemy. On October 20 (Sunday) the French sent heralds to Henry announcing that they would fight with him before he came to Calais. Having crossed the Somme Henry turned north, passing through Athies and Monchy La Gache, to Doingt.

To-day the main road from Athies to Albert leaves Peronne a little on the left and this road Henry seems to have followed. Meanwhile the French army, which had advanced along the right bank of the Somme while Henry was going up the left bank, retired to Bapaume, and as the English advanced about a mile beyond Peronne, they found the roads already trodden by the French, and this discovery filled them with despair. This place must have been the hill that rises up to the top of Mont St. Quentin, where the road from Doingt joins that out of Peronne. The army then advanced towards Albert, then called Ancre, and quartered at Forceville and Acheux.

On October 23 Henry continued towards Lucheu, passing between it and Doullens, and lodged at Bonnières, while the Duke of York with the advance guard was at Frévent. On Thursday, October 24, Henry crossed the Canche, probably at Frévent, and marched to Blangy, and found the French army drawn up in front of him near Maisoncelles. On October 25, St. Crispin's Day, the Battle of Agincourt was fought, for the account of which we are chiefly indebted to Elmham, Chaplain to Henry, who was an eye-witness, and watched the fight seated on his horse in the rear of the battlefield. The details of the battle are not the concern of this article.

There is great discrepancy in the accounts as to the actual numbers of the forces engaged in this battle. It is estimated that the English force was about 6,000 men, and certainly did not exceed 10,000, while the French force is said to have been ten times as numerous. The English are said to have lost 1,600 men and the French 10,000. The heavy French losses are accounted for by the fact that the English were ordered to kill their prisoners owing to a false rumour that the French were about to launch a counter attack, after a number of their men had already surrendered.

There are very scanty references in the chronicles as to what happened to the killed and wounded. The English killed were put into a barn together with such arms taken from the French that could not be carried away and there burnt. An exception was made with the bodies of the Duke of York and the Earl of Suffolk, who were among the slain. Their bodies were boiled, so that the bones might be taken home to England. The Duke of Gloucester was wounded in the abdomen by a dagger thrust. The wound could only have been slight, for he recovered before the army sailed from Calais. No other mention of the fate of the wounded has been discovered. It is known that Henry had no wheeled transport, and it can only be imagined that the wounded who could walk made their way back on foot, while probably others were transported in country carts commandeered from the neighbouring farms. The French dead were left on the field, where later they were buried. On the day after the battle the English left for Calais, which they reached without molestation on October 29. Shortly afterwards Henry returned to London, where he received a tremendous ovation, a spirited account of which is still preserved.

Such is the narrative of the campaign, which has been compiled from various published sources.

The names of three medical officers who accompanied Henry on this expedition have come down to us, namely, Thomas Morstede, surgeon, and Nicholas Colnet, physician, already mentioned; also William Bradwardyn, surgeon. Of Thomas Morstede much is known, and has been recorded by Sir D'Arcy Power in "The Memorials of the Craft of Surgery." He was surgeon to Henry V and Henry VI, and probably also to Henry IV. He became subsequently a Sheriff of the City of London. He is said to have written a goodly book on surgery, but if he did so all trace of it has been lost. In 1422 he was sworn as a supervisor of surgery, together with one John Harwe, whom we shall meet again directly as the successful defendant in a suit for malpraxis. Morstede died in 1450, and was buried in the Church of St. Olave Upwell in the Jewry. By his will he left his apprentice, Roger Brynard, ten marks sterling and his surgical instruments.

Of William Bradwardyn next to nothing was known beyond the fact that his name appears in the roll of those present at the Battle of Agincourt, published by Sir Harris Nicolas in 1832, and that he was associated with Thomas Morstede in a writ issued by the King in 1416, in which the two surgeons were appointed conjointly to provide the necessary medical equipment for the next campaign. Recently, through the good offices of Mr. Thomas, of the Records Office of the Guildhall, three documents have been found which throw some light on the life of this surgeon. William Bradwardyn (or Bredewardyn as his name is sometimes spelt) appears to have been a citizen and surgeon of the City of London, for in 1404, in the reign of Henry IV, there is an entry in the Husting Roll of a grant to him of a tenement in "Fletestrete," in the Parish of St.

Brides. In 1411 there is another entry in which he and his wife Margaret convey the said tenement to John Sapurton, Warden of the Fleet Prison. It is clear also that Bradwardyn was a person of some standing in the surgical world of that time, for he is described in 1422 as Vice-Master of the enfranchised art of London Surgery at a time when Thomas Morstede was one of the masters.

The following document taken from the Plea and Memoranda Rolls of the City Records appears to be of sufficient importance to be given in detail. It is written in Latin, the translation of which has been kindly made by Sir D'Arcy Power.

CITY RECORDS. PLEA AND MEMORANDA ROLLS. ROLL A 52, MEMBRANE 5.

On the tenth day of December in the third year of the reign of our king Henry the sixth after the Conquest there came here before William Crowmere, Mayor, and the Aldermen in the Guildhall chamber of the City of London Master Gilbert Kymer, doctor of physick and Rector of the London doctors, John Sombreshete, inceptor in physick, Thomas Southwell Bachelor of medicine the overseers of the medical faculty of London; John Corby medical practitioner, Thomas Morstede, knight, one of the masters of the enfranchised art of London Surgery, William Bradwardyn, esquire, Vice-Master of the same, Henry Asshborn and John Forde, surgeons enfranchised in Surgery etc, and acknowledged that the following was their award in these words—In the name of God Amen.

We, Master Gilbert Kymer Doctor of physick and Rector of the London doctors, John Sombreshete, inceptor in physick, Thomas Southwell Bachelor in medicine overseers of the medical faculty of London; John Corby medical practitioner as well as Thomas Morstede, knight, one of the Masters of the enfranchised art of Surgery in London, William Bradwardyn, esquire, Vice-Master of the same, Henry Asshborn and John Forde surgeons enfranchised in Surgery, being chosen as arbitrators to compromise in a certain matter of an alleged mistake in the surgical treatment of an injury to the muscles of the thumb of the right hand lying between William Forest who was injured, the plaintiff, and John Harwe, enfranchised surgeon, John Dalton and Simon Rolf barbers admitted solely to the practice of Surgery, defendants, having carefully considered the merits of the case and fully understood it by the plain evidence of the parties and the faithful witness of the barber John Parker somewhere admitted to practice Surgery, as well as of other trustworthy persons, well knowing, strictly sworn and sufficiently examined about the course of treatment—Find that the said William Forest, plaintiff, when the moon was dark and in a bloody sign, namely under the very malevolent constellation Aquarius was seriously hurt in the said muscles on the last day of last January and he lost blood enormously even to the ninth day of February last past, the moon remaining in the sign Gemini—That the said Simon Rolf himself staunch the blood successfully at the beginning and that afterwards the said John Harwe helped by John Dalton, the aforesaid barber artificially arrested it when the bleeding had recurred six times with great vehemence from the aforesaid wound even to (syncope) and as if William Forest would die. And that on the seventh occasion William was thought to be in danger of death owing to the excessive loss and quickly deciding that he would suffer mutilation of his hand rather than death the said John Harwe with the express consent of the said William, who was thus bleeding, when other remedies had failed stopped the bleeding with the cautery, as beseemeth, and saved his life and freed him from the bonds of death. Wherefore we praise, we award and we decide that the aforesaid John Harwe, John Dalton and Simon Rolf individually by themselves and by any of them, especially John Harwe, acted well and surgically in what they did in the aforesaid treatment and that none of them made any mistake in any way in this matter. Wherefore we absolve them and each of them and especially John Harwe, from being impleaded by the same William Forest in the aforesaid matter

by imposing perpetual silence on the same William in this affair; moreover we find that they themselves are so free from the fault attributed to them and to any of them and especially to John Harwe, defamed maliciously and undeservedly, that as far as in us lies we restore to them unsullied their good name so far as their merit demands and deserves in this affair.

We further declare that any defect of the aforesaid hand, or the mutilation or the ugly scar, so far as our industry avails to decide it, is due to the aforesaid constellation or to some peculiar defect or injury of the said William owing to the original wound. This was done in the year of our Lord one thousand CCCXXVIII and in the second year of our king Henry the sixth after the conquest on the ninth day of June in the Chapter house of the Brethren of the Minorite order of London, etc.

[Punctuation, which was not in the original manuscript, has been inserted.]

#### APPENDIX.

- (1) Pleas of Land held on Monday (May 11) after the Feast of St. John before the Latin Gate A° 6 Henry IV. [1405]:—

Grant from William Balle, John Crassewell, John Pole, tailors, & William Lathum, cutler, citizens of London to William Bredewardyn, citizen & "surgien" of the said City, Thomas Rede, John Russell, co Hereford, Matthew Boure & Sir John Whyte, clerk, co Hereford, of a tenement &c in "Fletestrete" in the Parish of St. Bride's, which they had acquired by grant & feoffment from John Knyvett, son & heir of Sir John Knyvett, kt. The tenement was bounded on the E by the churchyard, on the W by the king's highway running from Fleet St. to the Inn of the Bishop of Salisbury, on the S by the said Inn, and on the N by the tenement formerly in the tenure of Andrew de Guldeford. Witnesses, in addition to the Mayor Sheriff and Alderman of the Ward, Thomas Duke, John Askewith, Reymund Standelf, George Cressey, Richard Walworth. Dated London 17 June A° 5 Henry IV [1404].

HUSTING ROLL 133 (66).

- (2) Pleas of Land held in the Husting on Monday [22 Feb] before the Feast of St. Mathias the Apostle A° 13, Henry IV [1411/12].

Grant from William Bredewardyn, citizen and "surgien" of London & Margaret his wife to John Sapurton, Warden of Fleet Prison, & John Morell of Scropton co. Derby, of their tenement &c. in "Fletestrete" in the Parish of St. Bride's, which they had acquired by grant and feoffment from William Sergeant co Hereford. The tenement was bounded on the E by the Churchyard, on the W by the lane running from Fleet St. to the Inn of the Bishop of Salisbury, on the S by the above Inn, and on the N by a tenement formerly in the tenure of Andrew de Guldeford. Witnesses, in addition to the Mayor Sheriff and Alderman of the Ward, being John Askewith, George Cressy, William Ball, William Lathum, John Trum. Dated London 21 Feb A° 13 Henry IV [1411/12].

HUSTING ROLL 139 (44).

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## 10 Gask: *Henry the Fifth's Campaign of the Somme*

Mr. C. J. S. THOMPSON said that with reference to the apparently few skilled surgeons who accompanied the English armies in the field in the fifteenth century, as far as was known there was evidence to show that the nobles who joined the armies included, with their retainers, if possible, a man who had some skill in attending to wounds and dealing with the sick. Such an individual would probably also be a combatant, but there was little doubt that these men gave aid to the skilled surgeons who, like Morstede and Bradwardyn, accompanied the armies. Mr. Thompson drew attention to an interesting picture in the Historical Medical Museum by Forestier, representing surgeons embarking for France with the English army of Henry V in 1415, with the necessary workmen for making and repairing surgical instruments. He said that Morstede was attached to the army with about a dozen members of the London Corporation of Surgeons. When the second expedition to France was undertaken, the Corporation was apparently unwilling to provide even twelve men to join the troops, so the King authorized Morstede to embark as many surgeons as were wanted, whether they were willing or no, and to press into the service, also, all the workmen necessary for the making and repairing of the surgical instruments.

## Section of the History of Medicine.

President—Dr. ARNOLD CHAPLIN.

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### Greek Medical Etiquette.

By W. H. S. JONES, M.A.

It would perhaps not be very inaccurate to say that among the ancient Greeks there was strictly no such thing as medical etiquette. Modern etiquette, medical and other, is enforced by strong external pressure, and a violation of it leads to loss of caste, and possibly to social and economic ruin. Etiquette may be merely the "unwritten law," but there are also unwritten penalties to terrify the offender.

In ancient Greece, on the other hand, external pressure, other than the statute law of the land, seems to have been non-existent. It is true that we still have in the collection of medical works known as "*Hippocrates*" a document, known as the *Oath*, in which the medical man promises to conform to a certain code of conduct. But we do not know whether all physicians took this oath, still less if any penalties attended its violation. But all the facts point to there being no penalty. There is for instance in the Hippocratic collection a treatise the writer of which describes without shame his efforts to produce abortion in a patient, an act of which the taker of the Hippocratic oath swears he will never be guilty. There is no recorded instance of a man being expelled from the profession for unprofessional conduct. Nobody, so far as we are aware, had anything to fear from a General Medical Council. The age of trade unions was not yet. Neither had public opinion any strong influence upon professional conduct. Another tract in the Hippocratic collection, the *Law*, remarks that the only penalty inflicted on sinning doctors was lack of respect, and that this was no penalty at all, as quacks made a living out of it. In other words, a man so lost to shame as to transgress the laws of medical morality had no further check; he forfeited the respect of decently educated people, but did not care about it, as quackery was as profitable as scientific medical practice.

Modern parallels are not far to seek. There is as big a fortune in quackery as in a Harley Street practice, and an unscrupulous man driven from the latter may confidently hope to recoup himself if he have the wit to advertise a patent medicine. He may have lost what most men prize above riches or above even life itself, but if honour does not keep a man straight, to live without it in affluence is no great hardship. The way of the transgressor is made as hard as possible, but he can often find a refuge from hardship in disreputable but lucrative practices. When we have expelled a practitioner

from the profession there are still avenues open to him; in the ancient world there was no chance of preventing his practising, and to violate the medical code scarcely did him any material injury at all.

But there were forces at work in the ancient world which tried to keep up a high standard of medical honour. The charlatan was one thing, the respectable physician another. To what did these forces appeal? The answer is typical of the ancient—particularly of the Greek—mental attitude towards moral problems generally. Appeal was made to the artistic instinct. A true Greek loved his craft, whatever it was. For the honour of the craft he would do much that he would do for nothing else. "Love of the art," says *Precepts*, "and love of mankind go together." It will be said that such a sanction is but a feeble one, and incapable of resisting an appeal to the instincts of avarice or lust. That many would yield to the temptations is obvious, but we must not forget that the best sort of Greek was an artist first and a man afterwards. Nothing is so striking in the history of antiquity as the persistent refusal of the Greek to put sensual pleasure, or even physical comfort, before the satisfaction of the artistic sense.

It has been thought that besides this sanction a medical guild exercised control over practitioners, and forced them to observe a decent standard of morality. But there is no evidence that the Asclepiadæ, as they were called, could effectively control practising physicians, nor is there any reason to believe that the violation of the Hippocratic Oath was followed by deterring penalties.

There were only two deterrents, the law of the State and the doctor's love of his craft. Of the two the latter was by far the most important, the former coming into action only when some crime or serious wrong (murder, adultery, &c.), had been committed. To sum up, modern etiquette protects the interests of both patients and the profession, and is enforced by penalties of various degrees of severity exercised by the organized profession; Greek etiquette protected the patient where the law was not effective, and appealed to the artistic instinct without imposing penalties.

So far as I know, the only sources for Greek etiquette, with the exception of a few scattered allusions, are four small tracts in the Hippocratic collection—*Oath*, *Decorum*, *Law*, and *Precepts*. Considerable controversy has raged over the date of the first of these, but opinion is tending towards an Alexandrine or even later date for at least a great part of it. The other three have been strangely neglected, but I cannot imagine a competent scholar assigning to any of them a date earlier than the third century B.C. It seems, therefore, that for two centuries, from 500 to 300 B.C., during which period there flourished all that was best in ancient Greece, nothing was written, or at least nothing has come down to us, bearing on the question of medical etiquette.

In itself this is an interesting and possibly a significant fact. It may well be that during the really great period of Greek civilization, while the Hippocratic school flourished and the age of decadence had not set in, no need was felt to formulate a code of etiquette, just because it was in general instinctively obeyed, or perhaps because the big, general principle that a physician's duty is to help, or at least do no harm, to suffering humanity—so the great Hippocrates himself has expressed it—was considered a sufficient guide and check to control a practitioner's actions. Personally I am inclined to the latter view. It is typical of Greek thought at its best not to trouble about changing and non-essential details, but to lay down firmly first principles. Grasp these,



thought the Greeks, and all the rest can be derived by an intelligent man through the process of deduction.

The lack of references to etiquette in the earlier treatises of the Hippocratic collection and the insistence upon it in the later may be connected with the decline in the dignity of the profession. As the status of medicine fell less desirable characters became doctors, and those who cared for "the art," as it was always called, became desirous of raising the moral tone, and set down rules in writing. It is interesting to note that *Precepts*, the great repository of etiquette, is certainly very late, and was probably written by a Roman. Now at Rome medicine was a far less dignified profession than it was in Greece, practitioners in the ordinary sense being rare; medical treatment was usually the task of slaves. It is at least a strange coincidence that Dr. Singer, who has made a special study of the *Oath*, thinks that it, too, was compiled in its final state at Rome.

There is no Greek word exactly corresponding to the English word "etiquette." There is nothing unnatural in this, as ancient conditions were fundamentally different from modern. The unwritten laws, however, which the physician ought to obey were summed up in the word *εὐσχημοσύνη*, "that which is seemly and gracious," "decorum," "the behaviour of an artist and a gentleman."

I will now enumerate the acts which the *εὐσχήμων* was debarred from committing, and what course of conduct he was expected to pursue. Many aspects of decorum would now be criminal or at least civil offences, while others would be merely questions of good manners or ordinary decency. Herein lies one of the fundamental differences between ancient and modern etiquette: the former is far wider than the latter, and while its powers were less its duties were far greater. Ancient etiquette did not rely on the categorical imperative, but it embraced the whole duty of a physician.

(1) The physician should neither give poison nor sanction the giving of poison.

(2) He ought not to encourage abortion.

(3) He ought not to abuse his position to debauch any member of a patient's household.

(4) He ought not to give away information about the patient, whether that information is acquired in the course of his profession or in the ordinary intercourse of society. This last point is one to which I would draw particular attention.

(5) He ought not to advertise.

The following are the positive rules he should obey:—

(1) He should call in a consultant if in doubt or perplexity.

(2) He should be reasonable in his fees, and, if necessary, forgo them altogether.

(3) He should lead a pure and moral life, and should try to be, in the highest and widest sense of the word, a philosopher.

(4) He should avoid all ostentation in dress or manner, and try not to annoy the patient with unnecessary noises or odours, particularly those of wine.

I must remark here that there are a few points of etiquette that I have purposely omitted, as I hope to discuss them later; further, we cannot be sure that all these points of etiquette were a living force at one and the same time. Our evidence is far too scanty to allow us to be positive, though it is probable that they all gradually grew up together and came into vogue because of their obvious wisdom.

It should be noticed that while none of these clauses has as its object, or at least as its first object, the welfare and dignity of the profession, yet there is nothing startling in them; they are very much the kind of code a respectable physician would follow nowadays. Indeed in some respects the likeness between ancient and modern is sometimes almost ludicrous.

The ancients did not advertise by appeals to the eye, but by appeals to the ear. They delivered a speech or a lecture, just as on a lower level a cheap-jack holds forth at a fair. I was vividly reminded of a modern advertisement the other day on re-reading the following passage from *Precepts* :—

"If for the sake of a crowded audience you wish to make a display, your ambition is no laudable one, and at least avoid all quotations from the poets, as all such manifest feebleness of industry. I deprecate these far-fetched efforts, as the poetry is attractive only in and for itself. To use poetry as an advertisement of your skill is to imitate the labour of a drone, and to win the spoils of a drone."

In other words, poetry and sentiment have nothing in common with medicine or with the skill of a physician. The excellence of the poetry is no proof of the skill of the doctor nor of the value of the medicine. Thinking men saw through the fallacy as easily then as they do now, but the common herd were as easily duped in 300 B.C. as they are in 1923.

I would call your attention, however, to the fact that the ancient doctor was not *forbidden* to advertise; he was only warned that to do so was unseemly, and derogatory to the art. Every point of ancient etiquette proper is introduced by a "should" or an "ought," never by a "must." Etiquette was merely "good form."

Less blatant advertisements appear to have been not uncommon and more or less tolerated in spite of the rule of etiquette. There are still surviving inscriptions in honour of physicians who had given their services *gratis* to the state in times of emergency, and it is not taking too low a view of human nature to suppose that the generous physician was not blind to favours to come.

The temples of Asclepius, the relation of which to rational medicine is still somewhat doubtful, being in part hydropathic establishments and in part resembling Lourdes, were covered with advertisements. Grateful patients seem to have been compelled, or at least strongly persuaded, to put up inscriptions describing their cures, the modern analogue being the unsolicited testimonials with which we are so familiar in our patent-medicine advertisements.

There is one piece of etiquette—ancient etiquette I mean—which I have not yet mentioned. It is deeply interesting, but its history is shrouded in mystery and uncertainty. We know from Galen that in the Rome of his day it was considered unprofessional to operate. The Hippocratic *Oath* contains a clause in which the future physician promises to abstain from operating. This clause is supposed by some to be late, and indeed it is most unlikely that any such promise held good in the great period of Greek medicine. As far as we can tell from the Hippocratic writings, during this period (roughly 500-300 B.C.) physicians operated without fear or scruple. When did this sharp severance of surgery and medicine take place? We do not know for certain, and we can only guess. It was probably somewhere between 300 B.C. and A.D. 100. One thing is clear. No such cleavage could have taken place when the practice of medicine was in a flourishing and healthy state. The idea that there is something degrading in manual work is a sure sign of decadence.

I have long pondered over this problem, and tried to find a reason for the separation of medicine and surgery which should not be a slur upon both. Was it because of the difficulty of surgery? But it was not too difficult for the great Hippocrates, who mastered it and practised it with the most wonderful success. Was it because surgery was "messy" and disagreeable in pre-anæsthetic days? Again, the Hippocratics did not think so, nor would any one of sound medical opinion. Was it because of the dangers of operating in days when antiseptics were unknown? But the dangers were no greater than they had ever been, and danger would only justify a medical man in being careful and not operating unless otherwise death were a certainty. It would in no circumstances justify a promise never to operate.

I am aware that many scholars have supposed that the clause in the *Oath* only applies to one operation, that for stone, while others make it refer to castration, and not to operative surgery. I must point out that the Greek permits of neither of these views, which are also incapable of being reconciled with the statement of Galen. In other words, the historian is forced to admit that at one time between the two dates I have mentioned the medical profession had sunk very low, and certain duties were regarded as common and unclean, to be relegated to inferior members. It is indeed shocking to think that surgery was ever considered undignified.

*Εὐχρησισμὸν* told you what you *ought* to do, not what you *must* do under threats of pains and penalties. It is not surprising, therefore, that it included very much that nowadays would not be considered etiquette, but rather would come under the category of bedside manners. Some of our authorities go into this question with a wealth of detail which is sometimes rather amusing. Great stress is laid on politeness, urbanity, reserve, restraint in talk, gravity, neatness and sobriety of dress, and it is hinted that the practitioner should refrain from wine while making his rounds. He is to show calmness, authority and quietness, and in particular he is to speak about the patient and his illness as little as possible consistent with the necessary instructions to the attendants. By the way, I often wonder who these attendants were. Of course there were no professional nurses, and the nursing seems to have been done partly by slaves and partly by the pupils of the physician, medical students as we should call them, it being not unusual for the doctor to leave one of these behind to attend to the patient and carry out any medical orders. The physician was encouraged to be a philosopher, in the sense that his attitude towards his patients was to be serious, honest and kindly. The characteristics of a philosopher are thus given: disinterestedness, modesty, judgment, firmness, purity, strict morality, freedom from superstition.

One of the most important points of etiquette for an ancient Greek physician was the question of fees. In modern times there is a more or less regular scale of charges, and a patient can form a shrewd guess what an illness will cost him. As to ancient Greece, let us listen to the author of *Precepts*.

"This piece of advice is of importance. Do not begin by worrying the patient about your fee. If you do, you will arouse in the patient a suspicion that unless he agrees to your terms you will go away and leave him to his fate. So do not concentrate your attention on fixing what your fee is to be. A worry of this nature is likely to harm the patient, particularly if the disease be an acute one. Hold fast to reputation rather than profit. It is better to reproach patients you have saved than to distress men who are at death's door."—*Chapter IV*.

"Be not too harsh, but consider your patient's means. Sometimes give your

services for nothing, remembering a past service or your present reputation. If you can help a stranger who is in need, do your best for all such. For where the love of man is there is also the love of the art. Some patients are helped to recover from a serious illness by realizing that their physician is a good man."—*Chapter VI.*

We admire the spirit of the writer. At the same time it is easy to see that degrading scenes were not uncommon at the bedside of a sufferer. They were doubtless aggravated by the Greek's love of an argument, and his delight in haggling.

In one very striking case ancient medical etiquette was anti-social, and did not look to the good of the sick, but to that of the individual physician or surgeon. Nowadays all discoveries in surgery or medicine must be made public property. A new surgical method or an improved vaccine must not be patented. The principle underlying this rule is that if the novelty be bad, the patentee becomes at once a quack; if on the other hand it be good, the general welfare must override any pecuniary benefits which in other spheres of life would be the natural reward of the discoverer or inventor. Secrecy is condemned. But in ancient times things were different. Our informant is Galen, but as he is commenting on a passage in the Hippocratic collection, and explaining it, the practice he is referring to must be ancient. Moreover, although surgery is in question there is no reason to suppose that the rule did not apply to medicine. The custom I refer to is that of concealing as much of the body as possible during operations. Galen assures us that this was to prevent other practitioners from learning any secret methods the operator did not want them to know. The context does not imply the slightest condemnation; in fact the custom is considered quite fair and reasonable. It is unnecessary to point out that it was the natural outcome of the want of corporate unity and of an organized profession. The individualism of the profession in ancient times, while permitting or even encouraging a high standard of medical ethics in many respects, was no protection against the mercenary greed of the ordinary business man. The Greek did not fully realize that medicine is not a business but a profession.

The Hippocratic *Oath* forbids the physician to tell tales, and it is expressly stated that the rule applies whether it be in practice or whether it be in private life that the information is acquired. It is true that the saving clause is added, "whatever it is not lawful to divulge," implying that there were some things, learnt in practice and out of practice, which in certain circumstances might be made public property. We do not know what these exceptions were, and it would be idle to guess. But in two respects medical etiquette has changed since the time of the *Oath*: (1) Nowadays silence is restricted to facts learnt in the course of practice; (2) within these limits the rule has no exceptions.

The change is a most interesting one. In early times it was seen that reserve in these matters was wise, while at the same time some discretion was allowed. So long as the only sanction of etiquette was a sense of decorum such width of scope and such vague laxity of application was not only natural but almost necessary. But in modern conditions greater rigidity is essential. The violation of etiquette may bring serious consequences to the practitioner. Medical jurisprudence was unknown in ancient times, but is now a real force. The law of the land in many cases does not uphold the doctor's claim to keep silence. So it is felt that a clearer definition of the physician's duty is absolutely necessary. It is unfair—unfair to both practitioner and patient—to allow the former to exercise his own discretion. His way must be mapped

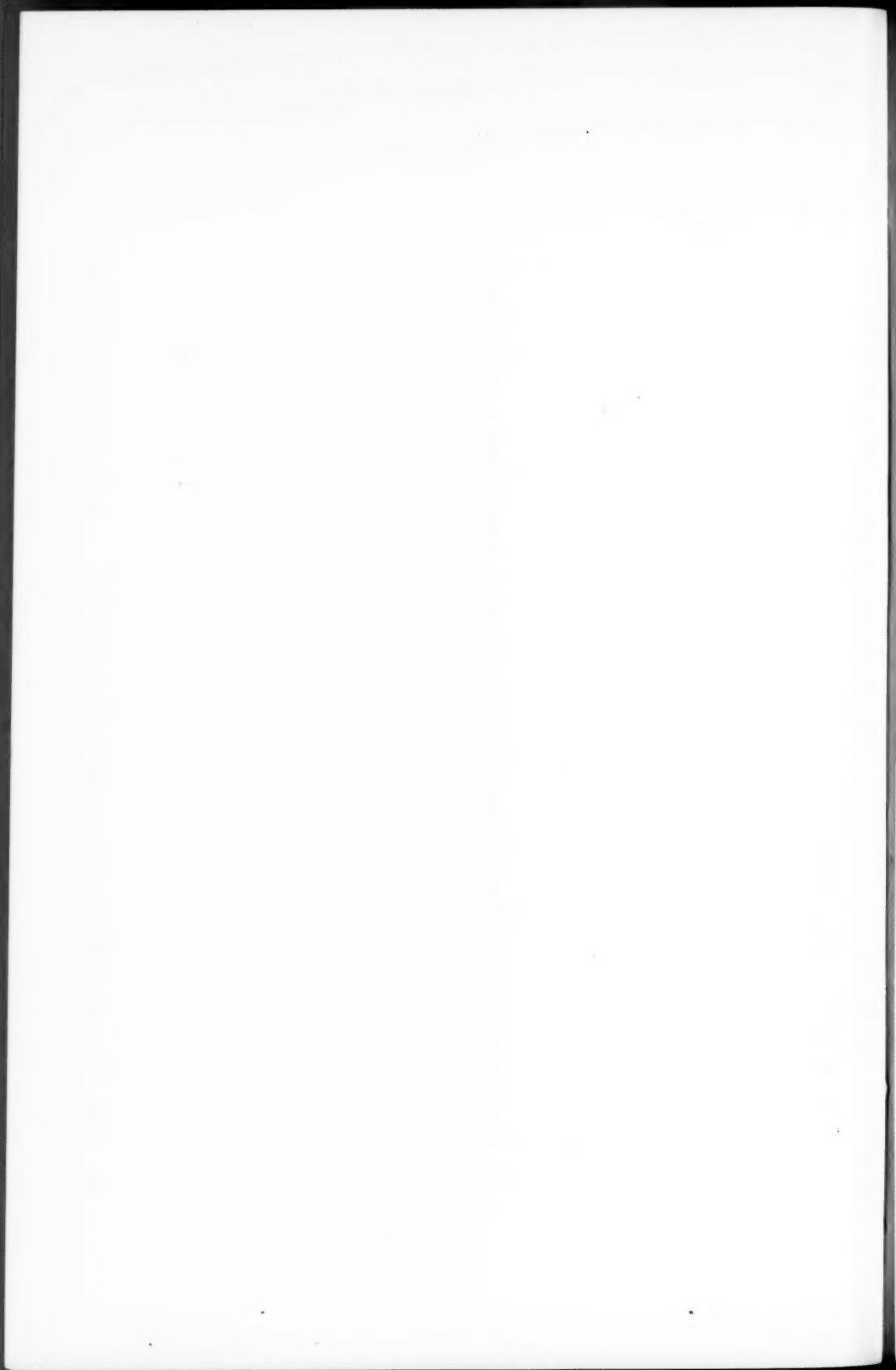
out for him, and he must be freed from the responsibility of deciding what he may divulge and what he may not.

I have tried in these rather random notes to lay stress upon the main differences between ancient and modern medical etiquette. They seem to me to converge on one point—in ancient times etiquette was a question of decency, of love of the profession, and the practitioner was at liberty to conform, or not to conform, at his own discretion. One of our authorities, the little tract *Law*, notices with regret the absence of external pressure. In modern times there is, besides this sanction, external pressure, applied in the name of the whole profession by the General Medical Council. The consequence of this difference is most important. It may, however, be summed up in one sentence: the legitimate practitioner is in one class, the quack is in another. Between the two there is a great gulf fixed. But in ancient times there was no such gulf. There was quackery and there was scientific medicine, but they shaded into one another. A man might be an excellent physician, but in part a quack, and his quackery would be condoned as a venial fault. Quackery, to a greater or less degree, pervaded medical practice except the highest manifestations of it, and scientific medicine leavened most of the quackery. The medical man was partly a quack, and the quack was at least very often in part a scientific physician. Of course at either ends of the line were pure quacks and purely scientific doctors, but between was a majority belonging neither to one class nor yet to the other. Again and again in our authorities we read protests against lack of long and thorough training, the implication being that an ignorant physician will be more of a quack than a scientifically trained man.

Such a state of affairs was bad for both the profession and the general public. The elimination of quackery is essential for the progress of medicine. A quack may do good by chance and accident, but ultimately quackery means harm and pain, and scientific medicine, in spite of its mistakes, means ultimately the conquest of disease and its consequences. The medical profession has no higher duty than the suppression of quackery.

Of course mere etiquette could not suppress quackery without a strictly guarded permission to practise, granted only after long training and severe tests. In ancient times all such tests were lacking. State doctors had to prove their qualifications, but so far as we know no tests were required before a man was allowed to treat private patients. There were no medical degrees. We learn from the *Oath* that at some period at least efforts were made to limit instruction in medicine to something that approximated to a guild; but there was nothing to prevent a man who did not belong to it from practising, if he could find patients.

This is only another example of the general laxity prevalent in ancient times in all things pertaining to the government of the profession. This laxity, as I have already stated, accounts for the width of ancient etiquette and for the only sanction that supported it, the respect and love of the craftsman for his art.



## Section of the History of Medicine.

President—Dr. ARNOLD CHAPLIN.

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### A Note on Thomas Davies, introducer of the Exploring Needle.

By ARTHUR T. DAVIES, M.D., F.R.C.P.

THOMAS DAVIES, the grandfather of the author of this note, was born in Carmarthenshire in 1792. He was educated at the London Hospital under the direction of his maternal uncle, Mr. Price, and began practice in the Mile End Road which was then on the borders of the country. After two years of practice his health gave way from an attack of hæmoptysis and he was advised to seek a southern climate. He went to Paris where he stayed one year, and then on to Montpellier, where after two years he took the degree of M.D. in 1821. Returning to Paris he worked under Laennec for two years at the famous Necker Hospital then at the height of its fame. On his return to London he started practice in New Broad Street, and on the recommendation of Dr. Billing, the additional office of assistant physician was created for him at the London Hospital. He was elected December 5, 1827, but did not live to become full physician. He was also elected physician to the Infirmary for Asthma, in 1825. This institution afterwards became known as the Royal Hospital for Diseases of the Chest. It was founded in 1814, and was the first special hospital started in Europe for the study of the diseases of the chest. It was also the first to start post-graduate lectures. Thomas Davies was elected a Fellow of the Royal College of Physicians on July 4, 1838. He died on May 30, 1839, after having been sixteen years in practice. In an obituary notice of him in the *Medical Times and Gazette* it is stated that "among the residents of the East End of London he went for many years by the sobriquet of 'The Man with the Horn,'" referring to the stethoscope of which he was one of the earliest users in this country. There is a short account of him by Sir Norman Moore in the *Dictionary of National Biography*.

Davies published his work on "The Diseases of the Lungs and Heart" in 1835. In it he gives his account and description of the *exploring* needle which he was the first to introduce as a means of diagnosis in certain thoracic conditions. In that work we read, pp. 344-348:—

"I have proposed an instrument by which you may prove the existence of the fluid with but little pain and no danger, it is the needle which I present to you; it is somewhat thicker than that used for acupuncture; it is about 1½ in. long, pointed like a



trocar and has a groove running nearly to its point; the groove should be made deep and as wide as the thickness of the instrument permits."

He then proceeds to lay stress on the importance of puncturing the centre of the intercostal space and goes on to say:—

"The needle being introduced to about three quarters of its length into the pleural cavity, determines at once the presence of the fluid and what is equally important to the future steps of the operation, its nature; for, if it be serous, it will readily pass along the groove and trickle down the back of the patient; if it be purulent and thick it will not flow freely, but a thick drop or two will be seen at the external orifice; and on withdrawing the instrument you will find the groove filled with pus. The different degrees of the thickness of the matter show also the size of the trocar you should subsequently use, as a small one is sufficient for the evacuation of serous fluid, but a large one allows the continuous flow of thick puriform matter. The advantage of this instrument is, that it demonstrates the presence of the fluid and allows, therefore, the future step of the operation to be conducted unhesitatingly, that it causes but little pain to the patient, and is as harmless as an acupuncture needle. Having withdrawn the needle you may then introduce a trocar, the size of which as I have already hinted should be according to the thickness of the fluid you have discovered; if it be mere serum, a small hydrocele trocar is sufficient, if it be thick puriform matter, a larger instrument must be introduced, otherwise the flow will be tedious. I recommend you to be extremely careful in having the instruments sharply pointed, for if they be not, after the pleura has been penetrated by them, the false membrane, from its imperfect attachment, may be driven before their point and the operator foiled. The quantity of fluid you should permit must be according to its thickness; if it be mere serum you may evacuate two or three pints and the lung will freely expand, if puriform I generally allow from 12 to 16 oz. to flow before I stop it—recollecting that the lung is then, to a certain degree, bound down by false membranes."

"It now becomes necessary to establish a communication between the pleuritic cavity and the exterior: for this purpose I fit a portion of gum elastic catheter, previously cutting off its rounded extremity, into the canula of the trocar, and pass it through that instrument into the chest, to the distance of about 3 in.; the canula may then be withdrawn, allowing about 4 in. of the catheter to remain at the exterior, which may be secured in its position by tapes attached to it, and passed round the body: the whole should be still further secured by adhesive plasters. The fluid now passes through the catheter instead of the canula of the trocar, and you may stop the flow by introducing a small plug, made of a portion of the bougie, into the external extremity of the former instrument. Having thus a free communication at will, I daily evacuate a certain quantity of fluid, retaining the catheter in the wound three or four weeks; in fact, until I can obtain no more fluid. You need not be apprehensive of the presence of the gum-elastic catheter in the cavity of the pleura. I have never seen it do harm. From its elastic nature it soon bends and places itself against the inside of the ribs."

"You will often find that after the second day sufficient ulceration is effected, by the presence of the catheter in the wound, to enlarge it; in that case the fluid flows, though very slowly, by the side of the instrument. I endeavour to favour this by directing the patient to lie on the affected side. Often, after the instrument is withdrawn entirely, a fistulous orifice will remain for some weeks, from which a slight discharge passes. I have always seen that this circumstance is favourable, and never observed danger to arise from it."

"Some have feared that air would enter and occasion inflammation. No doubt air does enter frequently into the cavity, but I have not perceived it produces this result; indeed, the slightest reflection must show us that the false membranes are as yet incapable of inflammation in consequence of their inorganic nature. Laennec states that the operation for empyema is rarely attended with success. I cannot agree with him in that opinion. If he had said that the operation, when performed for the cure of pneumothorax combined with effusion, was generally unsuccessful, I most fully agree;

for it has occurred to me, that, out of sixteen individuals in whom paracentesis has been performed for simple effusion of serum or puriform matters in the chest, twelve have perfectly recovered and four only have died. Where, however, there has been a combination of pneumothorax and effusion of fluid the operation has been invariably unsuccessful in its results. The causes which render the operation for simple effusion unsuccessful are, the existence of tubercular or other disease of the lungs, and above all, the thickness of the false membrane, which prevents the contracted lung from expanding. I have found that children most frequently recover, probably from the lungs being more commonly healthy in them, and from the great pliability of their chests, by which the parietes fall in more readily upon the contracted lung."

Thomas Davies was evidently closely intimate with Laennec as he gives the following account of him in his lectures, pp. 61-62:—

"Laennec very early obtained a reputation for scholastic acquirements, more particularly Latin and Greek. He was much attached to antiquarian pursuits particularly relating to the dialect of his own province, and nothing seemed to please him more than to meet a Welshman for the purpose of conversing with him and comparing the Armorican (Breton language) with the ancient British tongue. Laennec died of consumption, and I have no doubt that he long believed himself to be predisposed to that dreadful disease. You could not be with him even for a very short time, without seeing him expectorate, and examine the secretion with great minuteness; indeed, while I knew him, it was his constant habit. No doubt a consciousness of this kind led him to his investigations of pulmonary diseases."

Thomas Davies was deeply impressed with Laennec's discovery and studied the stethoscope carefully. He was the first to introduce to the British practitioner an accurate knowledge of the morbid anatomy of the lungs and heart distinguishable by the stethoscope sounds. On his return to England he gave a course of lectures in New Broad Street, on the value and use of the instrument. These were repeated at the London Hospital and were largely attended. They were subsequently published in the *Medical Gazette*, 1835.

In reference to Thomas Davies's nickname "The Man with the Horn," my attention has been drawn by Dr. Eric Bayley to an article in the *Lancet* of 1827, written by the French correspondent of that journal:—

"The patients labouring under diseases of the heart and lungs," he writes, "are sent to the wards of Laennec for the benefit of the auscultants or Laennec trumpeters as they have been maliciously called by some."

In the same number of the *Lancet* (p. 378) we read that—

"the treatment pursued by the French Professor is not likely to be adopted by any of our practitioners. He confined himself to simple expectation," a process which has been called "meditation on death."

Laennec was, we read,

"a believer in the doctrine of crises and critical days, hence he did not employ purgatives lest they should interfere with the salutary effects of nature. He pursued his expectant treatment waiting to see how nature should proceed with her operations, and if he could not catch her at her work he simply looked on—applied his stethoscope now and again—took a pinch of snuff and walked on."

In the *Lancet* of 1825-26, a review of a book by William Stokes, of Dublin, is headed by the quotation from Shakespeare:—

Hamlet: Will you play upon this pipe?  
Gent: My Lord, I cannot.

The review proceeds to remark scoffingly that the—

“physician has only to put his hand in his pocket, apply his mahogany oracle to his ear and presto, all was to have been plain to his understanding as if he had made an actual dissection of the parts themselves. How little did the venerable dispensers of health, who flourished sixty years ago, know to what profitable uses and ends they might have converted their indispensable bamboos. It remained for their successors to cut down these ambulatory superfluities to the more profitable dimensions of a pathological baton.”

## Section of the History of Medicine.

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### The History of Vaccination in Japan.

By Professor MIKINOSUKE MIYAJIMA, M.D.

(Kitasato Institute, Tokio.)

(Communicated by Sir GEORGE BUCHANAN, C.B., M.D., Ministry of Health.)

IN Japan, as in other Eastern countries, the practice of vaccination was preceded by the practice of "small-pox inoculation." This measure was introduced into Japan in the middle of the eighteenth century. It was not, however, until about 1820 that the first endeavour was made to give the country the benefit of Jenner's great discovery. In that year a Dutch scientist, Siebold, conveyed some vaccine lymph from Java to Nagasaki, but unfortunately the people were ignorant of the efficacy of vaccination and refused to give it a trial.

Twenty years later a description of Jenner's method found its way into Japanese literature and this was followed by renewed attempts to start vaccinations with imported lymph. Most of them failed because the vaccine lost its potency during the long voyage to Japan, but Takashima Shirodayu, the local governor of Nagasaki and a well-known scholar, was successful in vaccinating a few persons in Nagasaki and in Yedo (now Tokio) with lymph obtained from Holland. There were no arrangements, however, for continuing the vaccinations and the strain was soon lost. About the same time several Japanese physicians, notably Nagayo Shuntaso, Oyama Shisai and Inouye Sozui, began experiments with a view to obtain vaccine material by inoculating calves with the virus of variola. The experiments of Inouye Sozui were successful and the lymph obtained was distributed in some districts but unfortunately it was used in only a few cases.

Between 1845-50 Nabeshima Kanso, the feudal lord of the province of Saga and the intellectual leader of the movement for studying Western science, became an enthusiastic supporter of vaccination. Through the agency of his court physician, Narabayashi Soken, a supply of vaccine lymph from Holland was brought to Nagasaki by a Dutch physician named Mohnike in 1848. It had however lost its potency on the journey and failed to produce a successful result. Dr. Mohnike then sent to Holland for vaccinia "crusts." They reached Japan in 1849 and a successful result was obtained in one child out of three who were vaccinated with this material. From this case the child of an attendant at the court was successfully vaccinated. Lord Nabeshima then gave orders for the vaccination of his daughter and his adopted son Junichiro. In each case perfect vesicles developed and the lymph from them was then distributed in Yedo, Kyoto and other districts. At the same time a pamphlet

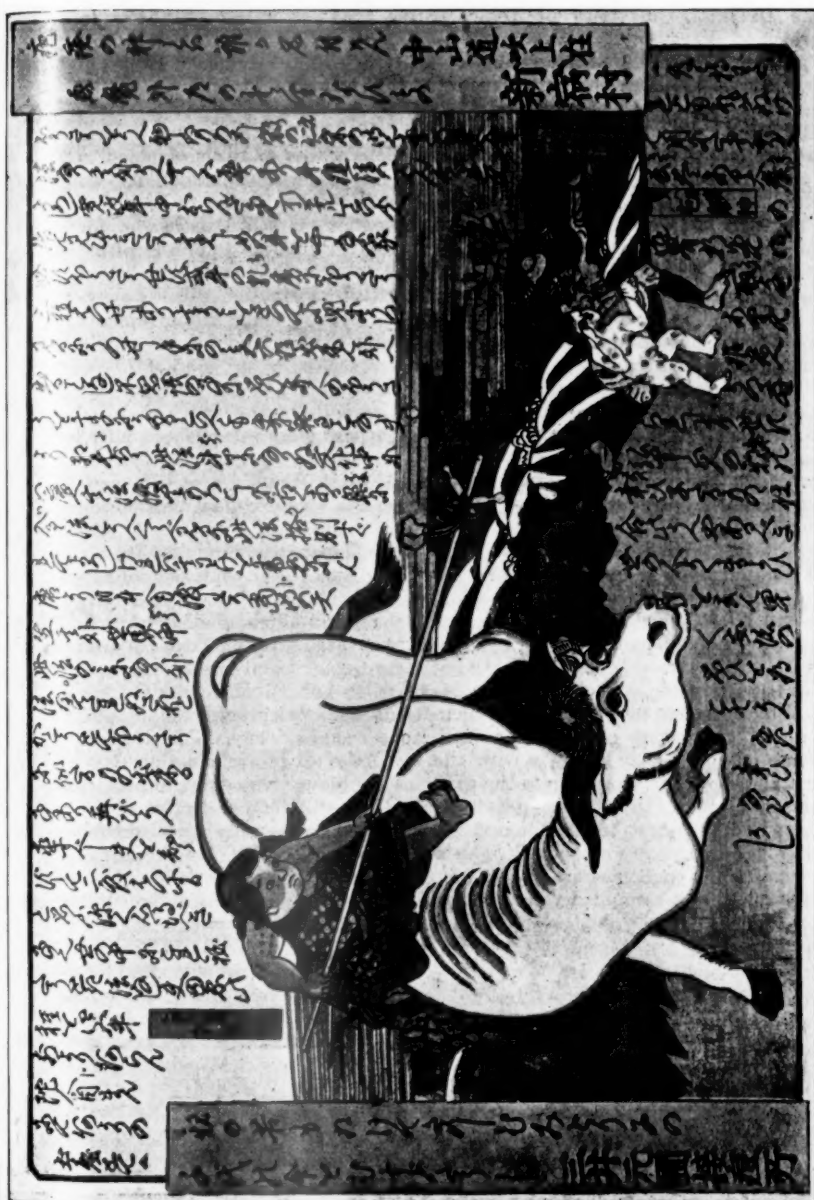
giving a general account of vaccinations was prepared by Dr. Narabeyashi at the request of Lord Nabeshima and widely distributed. It was entitled, "A Brief Account of Calf Vaccine," and is believed to be the first published report on vaccination in Japan.

Lord Matsudaira, governor of the province of Fukui, was associated with Lord Nabeshima in petitioning the central Government to obtain a supply of vaccine lymph from abroad, and when the successful results described above became known he made arrangements for distributing lymph and for carrying on vaccination in his province. The official support of these two public-spirited governors, combined with the efforts of physicians, overcame some of the difficulties of maintaining and transporting supplies of lymph and difficulties of communication under the feudal system, so that within a few years vaccination became a general practice in many parts of the country. The chief vaccination centres first established were at Saga, in Kyushu, and at Kyoto, Yedo (Tokio) and Fukui, in Nippon.

Serious obstacles to progress still remained. Among them not the least was the prejudice of practitioners who followed the teaching of the ancient Chinese Schools of Medicine. The central Government of the day was easily persuaded to believe that the introduction of methods and practices based upon Western learning would be prejudicial to the best interests of the country. As a consequence a decree was issued in 1848 prohibiting the translation and publication of all medical and other scientific works in the Dutch language, which was at that time the only medium through which Western knowledge reached Japan. It was a very curious coincidence that the year in which Dutch learning was prohibited was the year in which Mohnike brought vaccine lymph to the country, and it is probable that if those efforts to introduce vaccination had not been successful Dutch learning would have been entirely suppressed in Japan and the progress in the direction of modern civilization would have been greatly retarded. It happened, however, that even those who objected most strongly to the introduction of Western science could not dispute the efficacy of vaccination and the great benefit which the country derived from it. Therefore the Government wisely permitted vaccination although it was a practice brought in from a foreign country, and its subsequent universal adoption and popularity gradually led to a mitigation of the severity of the measures for prohibiting Western learning. In any history of the progress of Western civilization in Japan the influence exerted by the introduction and spread of vaccination should be particularly noted.

Among the arrangements made for popularizing vaccination may be mentioned the use of pictorial circulars. One of them, published and widely distributed by Kuwada Rissai, a vaccinator in Yedo, pictures a vaccinated child mounted on a cow and hunting down the devil of small-pox, and as a contrast another child not vaccinated and piteously crying in the clutches of the small-pox devil (*see figure*). The picture is accompanied by a popular poem and explanation, indicating a knowledge of the methods of skilful advertisement.

Between 1850 and 1860 many clinics, where vaccination was practised, were started in various parts of the country and they served also as the meeting-place of students and teachers of Western science. The clinic established by about eighty students and teachers in Yedo in 1859 was rebuilt on another site in 1860, and was brought under Government control as a central vaccination station. Later it became the chief educational institute for the study of Western medicine and the foundation of the present medical faculty



Translation of the first poem on the right:—

Who ever called him the "God" of Small-pox—  
The incarnation of a demon accursed?

Translation of the second poem on the left: Blessed be

(the vaccine) that relieves the parent from anxiety and brings to the happy babe the  
fruit of long life.

The writing in the picture is an account of the introduction of vaccine from Holland into Japan. It advises every parent who has a child to ignore his superstition and accept the treatment which will bring happiness to the family. The picture represents a child being tormented by the Demon of Small-pox. A healthy vaccinated child is riding on the cow which gave the vaccine.

of the Imperial University of Tokio. After the Restoration, in 1871, a "Bureau of Vaccination" was established in the Imperial University for regulating and controlling the distribution of lymph and vaccination work in general, and later, its duties were transferred to the Bureau of Medicine in the Government department of education.

Until 1873 the vaccine lymph used was derived solely from arm to arm operations, but in that year the Hon. Nagayo Sensai, who had been deputed to study the methods of preparation of vaccine in Europe, established at Tokio an institute for the preparation of calf vaccine. In 1875 the Bureau of Medicine was transferred from the Department of Education to the Department for Home Affairs, and its title was changed to that of "Bureau of Public Health." Vaccination was one of the subjects transferred to this Bureau, which issued in the same year regulations making compulsory the vaccination of all newly-born babies and re-vaccination between 5 and 7 years of age. Improvements were also made in the arrangements for preparing vaccine and for its supply throughout the country.

In 1896 the regulations relating to vaccination were revised and it was decided that the central Government should undertake the preparation of vaccine. Two Government institutes for this purpose were established, one at Tokio the other at Osaka. In 1899 the latter institute was closed and in 1905 the work of the former was transferred to the Kitasato Institute for Infectious Diseases which has since prepared all the vaccine required for distribution throughout the country.

The widespread employment of vaccination in Japan has brought about a great diminution in the prevalence and severity of small-pox. History tells us that during ten centuries prior to the introduction of vaccination there were sixty-five great epidemics of small-pox and that five emperors died from it. There have been epidemics since vaccination was begun, particularly between 1886-91 and in 1896-97, but they have been less frequent and less severe; and it has been possible, especially in recent years, to bring them quickly under control by measures for compulsory vaccination. This was exemplified in 1907, when the importation of small-pox into Kobe seemed likely to lead to an epidemic comparable in severity with the epidemics of pre-vaccination days. I must add, however, that despite the existence for many years of a compulsory vaccination law in Japan, the people in general are still very neglectful of the measure and it is easy to find among the poorer classes many unvaccinated children. For this reason, as well as because Japan is situated near countries where small-pox epidemics are frequent, it is highly important for us to enforce the law relating to compulsory vaccination as strictly as possible.



## Section of the History of Medicine.

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### The Black Death in England and Wales, as exhibited in Manorial Documents.

By WILLIAM REES, M.A., D.Sc.

(University College, Cardiff).

THE Great Pestilence has aroused much interest since the time when Professor Thorold Rogers ascribed to it the chief cause of the agrarian revolution of the fourteenth century, a revolution which, by breaking up the manorial system and releasing the serfs, marked the beginning of the end of mediaevalism in England and laid the foundations of modern individualist society.

Though discussion has been full, the literature on the subject is not rich. There is no one work in English which can be regarded as authoritative in all respects. Cardinal Gasquet's great work has never been superseded but that work is based upon evidence drawn mainly from ecclesiastical sources. Jessopp, in his "Black Death in East Anglia," relied almost entirely on the Court Rolls for his material. One-sided evidence of this character is naturally incomplete and only within recent years have the various fiscal documents of the manors been compelled to give of their spoil. The evidence of these documents, though calm and cold, is convincing. But results from these sources can only be obtained by patient search and comparatively few areas have as yet been examined in detail. Enough, however, has been found to justify a complete denial of the main thesis of Professor Rogers and to enable us to form a more accurate estimate of the Pestilence and its effects. This may be regarded as the position reached to date.

The disease is supposed to have begun in China as early as 1333. During the years 1347, 1348, rumour was passing through Europe of a great mortality which had come from China and India, by way of Persia and Mesopotamia, to the Saracen lands and the Tartar camp, inflicting such havoc that there was left among them "scarcely the tenth man or the tenth woman."<sup>1</sup> The Roman court<sup>2</sup> was informed that 8,000 legions had died among the pagans. This, said the Christian West, angry at the failure of the Crusades, could only be the penalty of unbelief. This idea was indeed taking hold among the Saracens themselves who "purposed verily to be Cristen but when they wist that the pestilens had now reached the Cristen men, their good purpose ceased and

<sup>1</sup> Chron. J. Capgrave, s.a. 1348.

<sup>2</sup> Chron. H. de Knighton, pp. 59 *et seq.*

they returned to their vomit."<sup>1</sup> The king of Tharsis, after a journey of some twenty days to be baptized by the Pope, turned back when he found that the death had passed beyond the Levant<sup>2</sup> and, "following the course of the sun," was making its way through Christian Europe into South Russia, into Balkania and by way of the Mediterranean ports into Italy (1348) and South France, following the various routes inland. Italian writers of the time suggest that the infection was conveyed to Constantinople from South Russia and was brought from there by Genoese ships to Italy but such a cosmopolitan city as Constantinople was naturally open to the disease.

Italy, hot and malaria-ridden, was sadly devastated, 70,000 inhabitants dying at Siena, 40,000 at Parma. Hardly a single inhabitant survived at Trapani, in Sicily. Boccaccio has left a vivid description of the terrible ravages of the plague at Florence.<sup>3</sup>

By the beginning of 1348 the plague had descended on the Papal city of Avignon, a town of about a hundred thousand inhabitants. The death-rate rose rapidly from 100 to 500 and to 1,000 a day at least. The English chronicles give detailed statistics for the Papal city. On one day 1,312 persons died and on another more than 4,000.<sup>4</sup> Sixty-two thousand people died there in three months. Other parts of Provence were similarly ravaged. The plague began at Marseilles at the opening of the year 1348, 57,000 people dying in a month. At Montpellier only seven out of 147 Preaching Friars survived.<sup>5</sup> Thence the disease spread westward into Spain and northward by the Rhone Valley into Central and into North-western Europe, probably being carried also by sea. Paris, at this time a town of about 275,000 people, was burying its dead at the rate of 1,300 a day, or, as the Chroniclers express it, "the living were not sufficient to bury the dead." From North France it spread to the Low Countries, to Germany and the North. In fact every country shared in the catastrophe.

Gasquet, following the account of the Malmesbury chronicler, Geoffrey le Baker and others, says that the epidemic entered England before the autumn of 1348 through the port of Melcombe (Dorset)<sup>6</sup> but it would be difficult to confine its entrance to one particular port. Certain chroniclers suggest Southampton,<sup>7</sup> another Bristol, another that it first occurred in the North of England and worked southwards,<sup>8</sup> another that it began both in the North and in the South.<sup>9</sup>

Undoubtedly the southern parts were affected from North France. The manor of Woppingthorne, in Sussex, was able to pay only one-fifth of its former rents.<sup>10</sup> The chroniclers are on the whole better agreed that the disease appeared at Melcombe during July or August and the epidemic early affected

<sup>1</sup> Chron. J. Capgrave, S.A. 1348; Chron. T. de Walsingham, I, 272.

<sup>2</sup> Chron. H. de Knighton, S.A. 1348.

<sup>3</sup> Gasquet, "Black Death," p. 23 *et seq.*

<sup>4</sup> Chron. H. de Knighton, S.A. 1348.

<sup>5</sup> Chron. H. de Knighton, S.A. 1348. "Of Preaching Friars in Provence there died 358 in Lent; . . . at Magdalena seven remained out of 160; at Marseilles only one out of 140 Preaching Friars and ten Friars Minor. Sixty-six Carmelites died at Avignon, and of the Hermits of England none remained there."

<sup>6</sup> R. de Avesbury. (Began at Dorchester about August 1, 1348.)

<sup>7</sup> Chron. H. de Knighton, S.A. 1348.

<sup>8</sup> Chron. J. Capgrave, S.A. 1349.

<sup>9</sup> Chron. T. de Walsingham, i, p. 272 *et seq.*

<sup>10</sup> Ing, P.M., C.E. III, 110 (11).

the south-western counties, Bristol and the small coast towns.<sup>1</sup> By the beginning of August, most of the tenants of Frome Braunch in Somerset were dead and there were other deaths in North and South Cadebury.<sup>2</sup>

Soon the plague is seen moving towards the Thames Valley. In Wiltshire all the bond tenants of Westdene and East Grimstead were dead before September, 1349. At Newbury (Berks.) all the free tenants died so that the rent of 40s. was not paid and there were no perquisites of courts.<sup>3</sup> All the villeins and cottars of Bokeland died and in the manors of Morton and Crookham there were several deaths among the bondmen. Oxfordshire seems to have been very seriously affected. At Hethe 21 out of 27 villeins died before Michaelmas (1349), at Staunton Harecourt 7 out of 13, at Cudelyngton 6 out of 14. There were also deaths at Ewelme, Burcestre and Marshbaldynon (5 villeins).

About Michaelmas 1348,<sup>4</sup> London was attacked, the disease lasting until the following August.<sup>5</sup> It is said that scarcely one-tenth survived, animals as well as men being affected,<sup>6</sup> but accurate evidence is not easily obtained though new cemeteries were opened in East Smithfield and on the site of the Charterhouse, also near St. Bartholomew's Hospital.<sup>7</sup>

By this time the plague was spreading into the Eastern Counties and the Midlands and was at its height in the summer of 1349. The bondmen and cottars of the manors of Caynho, Clophull and Ampthill (Beds.) all died,<sup>8</sup> also at Salden (Bucks.) and half of the rents of Great and Little Kimble (Knebrell), Weston Turvill and Hoggeston could not be levied.<sup>9</sup> At Wappenbury (Warwick), at Eston, Torpell, Upton, Westwarden, Earls Barton and Grafton (Northants), there was considerable decrease in the rents but in other parts, e.g., in the borough of Maldon, the losses were slight.<sup>10</sup> Further north, the manor of Wissinden (Rutland) lost nine of its villeins and the only bondman of Seyton died; the rents of Great Wyrleye (Staffs.) were reduced from £5 to £3 owing to pestilence and poverty.<sup>11</sup> Similarly, there were reductions in receipts at Greatham (Lincs.) also Wodethorp and Pulterton (Derby). Dr. Jessopp says that 2,000 clergy must have died in the diocese of Norwich within a few months. He further quotes the case of Hunstanton where 78 people died in two months, 172 in eight months. Henry of Knighton says that more than 380 people died in St. Leonard's Parish in Leicester, 400 at St. Cross, 700 at St. Margaret's.

Yorkshire seems to have suffered severely, especially the districts of Holderness and Cleveland, and the "Inquisitions" of this date contain numerous entries for that county. There were reductions in the rents received at Cotyngham, Wyveton, Kirkby, Moresheved, Aton, Hemelyngton, Cropton, Yapum, Castellevyngton, Tampton, Neuby, Kildale, Darthynton, Emeley, Sprotburgh, Cathwayt, Scorbey, Sutton, and Skelton (Cleveland). Osgodly was badly attacked, also Bulsham (Yabeton), all the tenants here falling victim

<sup>1</sup> Eulogium, Chron. (Rolls S.).

<sup>2</sup> Ing, P. M., C.E. III, 104 (27).

<sup>3</sup> *Ibid.*, 101 (3).

<sup>4</sup> Chron. R. de Avesbury, s.a. 1348 (about November 1).

<sup>5</sup> Bermondsey Annals, s.a. 1348.

<sup>6</sup> Cf. Walsingham, s.a. 1349; Capgrave, s.a. 1349; Bermondsey.

<sup>7</sup> R. de Avesbury, s.a. 1348. (About Michaelmas, 1349, the Flagellants arrived from Holland.)

<sup>8</sup> Ing, P. M., C.E. III, 98 (8).

<sup>9</sup> *Ibid.*, 98 (9).

<sup>10</sup> Ing, P. M., C. E. III. 99 (5). Maldon—six cottages idle.

<sup>11</sup> *Ibid.*, 103 (18).

and the rents (20s.) were lost. At Eastbraume the perquisites of the courts amounted to 2s. only. At Middleton, 12 bovates of the *naviti*, at Kebelyngcotes 10, at North Dalton 14, at Sledmere 40, at Wynstow 11, were by 1350 either waste or rented out. At North and Eastgaveldale there were 9 bovates waste. Conditions were similar at Hundemanby, Siwardby and Ruddeston, 10 bovates out of 14 being idle in Bruyngton.<sup>1</sup> Further north the border counties were in parts severely attacked though in others there are not the same signs of devastation. The Scots sought to take advantage of "the foule deth of Engelond" to invade the northern counties, but they, too, fell victims to the disease and carried it to Scotland.<sup>2</sup> Le Baker is of the opinion that the uplands of Ireland were spared until 1357, but Gasquet makes it clear that Ireland also suffered.

The plague seems to have entered South-east Wales from Bristol. By March, 1349, it was particularly destructive in the lordship of Abergavenny.<sup>3</sup> The hamlets of that manor yielded on the whole about one-third of their former rents, some being less affected than others. The rents of assize of Werneryth fell from £13 10s. to £1 14s. 6d., and those of Trefgoythel from £3 10s. 6d. to 6s. The surrounding lordships of Usk, Monmouth, &c., were not affected to this extent, but the losses in the neighbouring lordships of Brecon, Huntington and Glamorgan were very slight.<sup>4</sup>

The borderlands of Worcester, Hereford and Shropshire were stricken more or less severely. The potters of Hanley (Worcester), who used to pay 13s. yearly for clay, were all carried away. At Havercote the rents fell from 10s. to 3s. 4d. In the lordship of Caus (Salop), the rents of the freemen of one of the hamlets fell from £8 to 30s.; in Colmere and Hampton from £4 to 10s.; in the hundred of Bradefud from £16 to £8, but in other places the "decays" are comparatively small, e.g., at Schelve from 36s. to 26s.; at Wyleley from 20s. to 12s.<sup>5</sup>

The records of the lordship of Ruthin<sup>6</sup> are interesting. Before the pestilence the usual few deaths are regularly recorded but during the time of the plague the death-rate rose rapidly, as will be seen from the table given (p. 31). It is clear that during these months there were two periods of great severity.

Farther west the mountain region of Snowdonia, especially the Isle of Anglesey, was overrun, certain of the hamlets suffering heavy mortality but others apparently were little affected and even entire commotes escaped lightly.<sup>7</sup> Again, in West Wales the track of the epidemic is discernible here and there but inasmuch as the communities paid their rents collectively, it is difficult to determine the full extent of the ravages among the freemen. In the bond villages, however, the effects are clear. Many of the bondmen fled, and at Llanllwch, near Carmarthen, the twelve villein tenants died. In fact, the whole district of Carmarthen suffered seriously, the disease probably coming by sea, for even as early as March, 1349, the two Collectors of Customs of the King's Staple at Carmarthen had fallen victims.<sup>8</sup>

<sup>1</sup> *Ibid.*, 113 (25).

<sup>2</sup> Chron. G. le Baker, p. 99 *et seq.*; H. de Knighton, s.a. 1348; Chron. T. de Walsingham, 1349. "God and St. Mango, St. Romayn and St. Andreu, scheld us this day for Goddis grace and the foule deth that Ynglessh men dyene upon."

<sup>3</sup> See Appendix I, Abergavenny.

<sup>4</sup> Ing, P. M., C. E., III, 101 (5).

<sup>5</sup> See Appendix II, Caus; Appendix III, Chester and Flint.

<sup>6</sup> Court Rolls, Portfolios 217, 218, 219 *et seq.*

<sup>7</sup> See Appendix IV, Lordship of Denbigh; Appendix V—(a) Anglesey and (b) Carnarvon; Appendix VI, Montgomery.

<sup>8</sup> Min. Aces. Bundles, 1158, 1218.

S.A.  
1348  
Anglesey Chron.  
Rolls Ser. 92.2  
p. 62

Rolls Ser.  
28.1.1.410

S.A. 1379

It is clear from the foregoing that districts and even villages suffered unequally. This is confirmed by other evidence. Thus, the Bishop of Winchester's manors immediately east and south of Winchester, e.g., Cheriton and Twyford, had heavier losses than those further south at Droxford and Bishop's Stoke which were almost untouched. Indeed, the deaths from the epidemic on the estates of the Bishop are in the main insignificant when compared with those experienced in certain parts of the country.<sup>1</sup> It seems that the darkest parts on the map would include Sussex, the Bristol neighbourhood, the Upper Thames Valley (particularly Oxfordshire), Monmouthshire, Shropshire, North Wales and Yorkshire.

TABLE TO ILLUSTRATE THE COURSE OF THE PESTILENCE OF 1349 IN THE LORDSHIP OF RUTHIN. YEAR 1349.

Court	March 12	March 25	June 11	June 29	July 20	August 10	August 20	Sept. 21	Oct. 9	Oct. 23
Colyan ...	...	...	—	10	25	8	44	12	Several	Several
Llanfair ...	—	1	1	13	29	14	45	7	Several	Several
Dogg ...	Nov. 25 1348-1	1	...	25	11	13	20	9	3	2
Abergwillar ...	...	1	7	14	4	2	19	1	—	—
Ruthin Town	2	...	...	77+	14	17	44	4	Few	Few

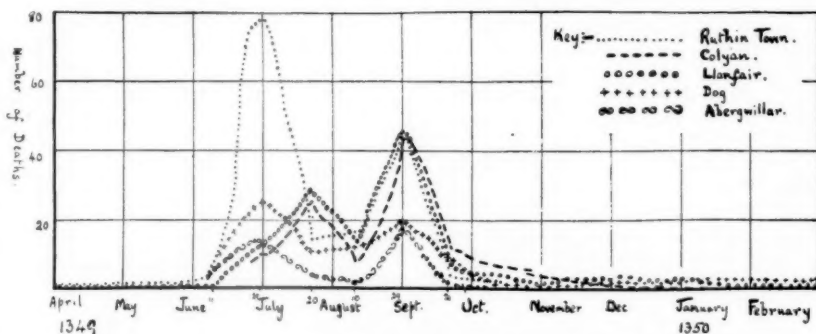


Diagram to illustrate the course of the Pestilence in the Lordship of Ruthin, 1349.

During the autumn the plague subsided but in Ruthin several deaths continue to be recorded in certain parts of the lordship during October and even into January and February of 1350. The death-rate there continued well above the normal during succeeding years, 1 death being definitely attributed to the Pestilence in 1351, about 9 in 1354, 2 in 1357, 2 in 1358, 2 in 1359, 1 in 1360, in addition to other deaths. During the autumn and winter of 1361-2 there was again a rapid increase, later termed the Second Pestilence, which subsided in the following spring. During succeeding years only few

<sup>1</sup> Oxford Studies, v, Levett and Ballard, "The Black Death."

deaths occur until the late summer and autumn of 1369 after which time there are only slight traces.

The English Chroniclers make reference to these later outbreaks, e.g., that of 1361 which was supposed to kill off men rather than women;<sup>1</sup> that of 1369 which was accompanied by losses among the animals;<sup>2</sup> that of 1375, when, as a result of the intense heat, there was a considerable outbreak among both men and women;<sup>3</sup> that of the summer of 1379 when there was a great pestilence in the north of England, "that nothing like it was seen before so that the whole 'patria' was suddenly deprived of its strongest men";<sup>4</sup> that of 1389 when there was a pestilence at Cambridge, men dying of madness;<sup>5</sup> that of 1390 and 1391 when there was great mortality of children, especially of boys;<sup>6</sup> that of September, 1393, when there was an outbreak in Essex.<sup>7</sup> There was also a general plague in 1407<sup>8</sup> and again in 1413.<sup>9</sup>

The outbreak of 1361 was usually regarded as the Second Pestilence and that of 1369 the Third. These two visitations were severe in South-east Wales especially the Monmouthshire district. Very heavy losses in rents are recorded at such places as Llantrissant, Trelech Town and Hodenach Manor (Monmouth). By 1362, 1,171½ of the 1,313½ "works" of the customary tenants were in "decay" at the Manor of Caldecote,<sup>10</sup> four of the tenants dying in that year. By 1366, 3 "semi-virgaters," 26 "quarterers," 6 "gafolmen," 3 "tenants semi-quarterers," 10 cottars together with certain holders of assort were dead. By 1372 that number had increased to 4 "semi-virgaters," 28 "quarterers," 7 "gafolmen," 2 "semi-quarterers" (= 4 tenants), 14 cottars, leaving the manor almost devoid of its bond tenants. In this case the number of survivors compares with the chroniclers' estimates of one in ten. But it must be remembered that such a high proportion of deaths is exceptional and that in many parts few or no losses are recorded. Under the circumstances it would be difficult to attempt even an approximate estimate of the average loss. The Malmesbury chronicler placed such estimate at one-fifth<sup>11</sup> but in Wales the death-rate is in many places much higher than in the Winchester district where at Twyford the rents were reduced to three-fourths and at Cheriton to five-sixths of their former amounts.

There had been plagues in England before 1348, e.g., in 1087 and in 1221,<sup>12</sup> but it cannot be determined whether they were of the same nature. The pestilence of 1316 took the form of dysentery accompanied by acute fever or throat affection (pestis gutturosa) with a rash (maculare) and came as a direct

<sup>1</sup> Worcester, S.A. 1361; Walsingham, S.A. 1361; Bermondsey, S.A. 1361.

<sup>2</sup> Worcester, S.A. 1369; Walsingham, S.A. 1369; Bermondsey, S.A. 1368.

<sup>3</sup> Walsingham, S.A. 1375; Worcester, S.A. 1375. [Fourth Pestilence.]

<sup>4</sup> Walsingham, S.A. 1379.

<sup>5</sup> Walsingham, S.A. 1389.

<sup>6</sup> Walsingham, S.A. 1390.

<sup>7</sup> Walsingham, S.A. 1393.

<sup>8</sup> Walsingham, S.A. 1407; see Royal Letters of Henry IV (Rolls S.), Letter 109. Letter of Countess of March to Henry IV, time of the Glyndwr Rebellion (nodate), "and now the pestilence is so severe and cruel where we are that I am much afraid to die in great debt. By no treaty can we obtain sufferance from our enemies to withdraw ourselves to our fortress of Colbrandespath to remain there till the mortality has ceased."

<sup>9</sup> Walsingham, S.A. 1413.

<sup>10</sup> Min. Accs. 671/11,000 *et seq.*; 921/16; 1156/21, *et seq.*

<sup>11</sup> Eulogium, S.A. 1348.

<sup>12</sup> Ann. of Waverley.



result of the chronic underfeeding and bad food during the famine of 1315-16.<sup>1</sup> In this case, said the chroniclers, knowledge of physic could find no remedy as in the past, and medicinal herbs could give no relief, and, indeed, poisoned rather than healed.<sup>2</sup>

The plague of 1348, usually termed *The Pestilence*, *The Murrian*, *The Mortality* or *The Death*, is usually regarded as the bubonic plague in a particularly virulent form but this hardly explains all the symptoms as may be gathered from the accounts of experienced observers. The Continental accounts are more comprehensive than the English, and Gasquet has drawn largely from these sources. He also cites a modern opinion that the disease was at that time new and is still not extinct, as is shown by a plague in Turkestan, in 1892, with symptoms almost identical with those of the Great Plague.<sup>3</sup>

Among the general symptoms of the disease may be included, a dark-coloured carbuncle in the groin or under the armpits, a devouring thirst, the tongue dry and cold, the voice hoarse and jerky, the eyes haggard, the skin dark and moist, vomiting, and convulsions, from which the victim was only relieved by death.<sup>4</sup>

Symon Cauvin a doctor of Paris and Montpellier has left the following details in a Latin poem written in 1350, now in the *École des Chartes*.

"A burning pain, starting either in the groin or under the armpits gradually spread over the precordial region and the vital parts were attacked by a mortal fever. The heart and lungs were affected and the respiratory passages were choked with the poison. The strength suddenly declined and the patient could only survive a few days. Even the future victim could be distinguished by the paleness of the face. There seemed no refuge from this scourge, neither heat nor cold nor the fresh country air, the cold north or the warm south. So contagious was it that when the sickness commenced in a house, scarcely one escaped. The slightest contact, a single breath sufficed to transmit the disease. Those who tried to aid the sick fell victims. The ill-nourished were easily stricken. Those who lived a temperate life fared best. The number of the dead was greater than the survivors and cities were deserted, thousands of houses standing with open doors or locked up, their owners dead or fled."

Wm. de Nangis<sup>5</sup> speaks of the young people being in the main the victims. Some died suddenly. In the case of others, lumps appeared under the arms or in the groin, an infallible sign of death. Boccaccio refers to the futility of drugs to cope with the disease and physicians were helpless in its presence. Few escaped if once attacked, the victim seldom lasting more than three days. Animals as well as men were susceptible to the disease. Another writer speaks of a rapid fever, blood-spitting and ulcers.

Certain Welsh poets have made reference to the plague in Wales. Jeuan Gethin lost several of his children in the early fifteenth century.<sup>6</sup> In lamenting

<sup>1</sup> J. de Trokelowe, *Annals*, S.A. 1316.

<sup>2</sup> *Ibid.*; Walsingham, S.A. 1315.

<sup>3</sup> Gasquet, "Black Death," pp. 10-11 (note).

<sup>4</sup> See Ing., P. M., C. E., III (File 98). Halsham (Holderness), (April 1, 1350). "John le Conestable being of good memory but afflicted with great weakness for the four days preceding, at the time of the mortality then raging in those parts, languishing in *extremis* about that hour of day called Midovernone, made his charter concerning the said manor. He was of sound mind (sanus et incolumis)."

<sup>5</sup> G. de Nangis, "Continuatio Chronici," Soc. de l'Histoire de France. Gasquet, "Black Death," pp. 54-55.

<sup>6</sup> For these extracts, I am indebted to Mr. G. J. Williams, M.A., lecturer in Celtic in the University of Wales, Cardiff.



his two sons, he says, "I wept when I saw the black rash on his two arms." In another poem on the Plague, he says:—

"We see death coming into our midst like black smoke, a plague which cuts off the young, a rootless phantom which has no mercy for fair countenance. Woe is me of the 'shilling' in the armpit; it is seething, terrible, wherever it may come, a head that gives pain and causes a loud cry, a burden carried under the arms, a painful angry knob, a white lump. It is of the form of an apple, like the head of an onion, a small boil that spares no one. Great is its seething, like a burning cinder, a grievous thing of an ashy colour. It is an ugly eruption that comes with unseemly haste. They are similar to the seeds of the black peas, broken fragments of brittle sea-coal and crowds precede the end. It is a grievous disease that breaks out in a rash. They are like a shower of peas, the early 'ornaments' of black death, cinders of the peelings of the cockleweed, a mixed multitude, a black plague like halfpence, like berries. It is a grievous thing that they should be on a fair skin."

Gasquet quotes the testimony of the Eastern Emperor as to the pathology of the disease. Other ills, he says, paled before this, which treated weak and strong alike but affected people differently. Some died after an hour, others after a day, others in three days.<sup>1</sup> In this case, the disease opened with a fever, the patient losing speech and falling into a comatose condition, practically paralysed. Others were affected first in the lungs and breathing organs, the throat and tongue burnt with fever, black with blood. Spots broke out over the body and the victim tossed in an agony of pain. There was no remedy, and what gave relief to one was fatal to another. The patient immediately lost hope, but it is remarkable that some recovered and were then in part immune against further attack.

The above description by the Emperor would suggest that there was more than one disease raging at this time. Some writers are more explicit on this point. Thus one writer speaks of a three-fold infection: (a) An infection of the lungs and breathing passages; (b) lumps under the arms; (c) lumps in the groin. Geoffrey le Baker speaks of aposthumes (apostemata) breaking out in various parts of the body, so dry and hard that scarcely any liquid could be drawn from them. Certain persons by lancing these or by long patience recovered. Others had small black pustules covering the body and from this form of the disease few recovered.<sup>2</sup>

The Pope's physician, writing of the plague at Avignon, says that for the first two months the disease took the form of blood-spitting and fever from which people died within three days. During the remaining months the symptoms were carbuncles under the arms and in the groin, fever and possibly blood-spitting, both forms being highly contagious.

Attempts have been made to ascribe great and far-reaching effects to the "Death" of 1349, while in other quarters it has been the fashion to minimize those effects. It is the cumulative effect which is important. Changes far too sweeping and fundamental for the particular "death" of 1348-9 are not impossible as a result of a series of visitations extending over a period of twenty-five or fifty years. An adequate estimate, therefore, can scarcely be formed from a study of a short period or of a single district. Where the visitation was but slight, the damage could be easily repaired, and such a district would only be affected indirectly.

<sup>1</sup> Cf. Henry of Knighton, who in speaking of the plague at Bristol, says that few occupied their beds beyond three days.

<sup>2</sup> Chron., G. le Baker, pp. 99 *et seq.* (Cf. the description given by Jean Gethin above.)

It is hardly possible, however, within the scope of this paper to do more than indicate the trend of society. The fact that the pestilence coincided with a period of change makes it difficult to determine accurately the direct results of the "Black Death." Had the "Death" occurred half a century earlier, its place in the history of the time could more easily be stated. But changes, economic and social, were already appearing long before the "Black Death," changes which, though yet undeveloped, possessed great possibilities. An important feature in mediaeval society is the absence of initiative, economic and mental. The mode of life, the daily thought ran in traditional channels, hedged in by custom. By the late thirteenth century, certain elements hardly in keeping with this rigid conservatism may be detected. The serfs, who had hitherto carried out their services in detail on the lord's demesne in each manor, were beginning to make small payments in lieu of performing the services. This naturally gave less emphasis to their serfdom. Again, the principle of mere subsistence, the storing in summer of food necessary for winter, was giving way to a fuller life, to more business-like methods and the accumulation of wealth, as seen in the increase of luxury in the houses of the rich. The voice of criticism and new thought is beginning to disturb the simple piety and unquestioning belief of former days.

During the fourteenth century there is rapid development along these lines and the channel of the past does not suffice to contain the rising flood of new life. Individualism is taking the place of custom as the basis of society and the individual *motif* is becoming the mainspring of human activity. Society is fast losing its homogeneous character. The pursuit of wealth becomes an aim and the beginnings of modern capitalism are more perceptible. The manor is being worked for profit or instead of being a grouped unit, a single organism, it is let out in numerous individual holdings each worked by a "farmer" for his own profit, the lord thus becoming a rent-receiving landlord. With the gradual disappearance of the manor goes the system of serfage and manorial government as exercised through the courts of the manor. Trade is becoming more competitive.

In Wales the lowland manors were subject to the same influences as the English manors but the tendencies towards the complete break-up of the manor system are more marked especially as a result of the rebellion which later swept the country. In the lands of the Welsh the system of kindred holding was already in process of dissolution, especially under English influence, and rights in land were slowly passing to the several families of a kindred and to individuals. The "Black Death" assisted in this development. The escheats were withdrawn from the *tribal lands* to be transferred to the *feudal* and there were greater facilities for the transfer of land in general. By this and other means, direct tenure and individual responsibility were being introduced, thus bringing the Welsh lands on the same basis as the English and assisting in the union of England and Wales.

The way was rapidly being prepared for the central *State* of the sixteenth century when the new groups, landlords, tenant-farmers and workmen, become subjects of the national state and trade assumes national proportions. In this movement the epidemics of the fourteenth century played an important part.<sup>1</sup>

<sup>1</sup> See Henry of Knighton for some of the immediate effects of the Death of 1348-49. The decay of buildings in towns and villages, the fall in prices of foodstuffs and animals with rapid increase in the following winter, &c.: "There was lack of servants, for the lack of which, women and children were sent out to drive the ploughs and carts. . . . So great was the lack of servants and domestics that none knew what was to be done. . . . Meanwhile the king sent to each county that the reapers and other workers should not take more than they used to take, under penalty. . . . Magnates pardoned rent and services."

Nevertheless they did not originate the movement, but hastened it, thus helping to lay the foundations of modern society in England at an early date.

The change in thought was no less fundamental. The voice of criticism grew louder and more insistent and came to be directed against the rapidly-growing evils in Church and State, the evils of wealth and class, the evils of ecclesiasticism—a vague revolt against the new individualism as well as against the old mediaeval standpoint. Gasquet's theory that the death of priests on a large scale in 1349<sup>1</sup> and the opening of the profession to untutored clerks began the movement which ultimately led to the break with Rome, is unsatisfying and ignores fundamental issues.

The psychological effects of the plague were immediate. Like the recent War, the pestilence was sufficiently widespread to shake society to its foundations. The "Death" of '49, followed by the drought and storm of 1352, the cold winter and storms of '53, the appearance of the aurora in '55, the earthquakes of '82 and the famine of 1390 could not but be interpreted as divine intervention and the sense of impending destruction was abroad. Some turned to sorcery and astrology.<sup>2</sup> The horror and suddenness of the calamity, the consciousness of unseen forces roused all the primitive instincts. Isolation took the form of flight, abandonment of the sick and of the dead regardless of family or affection. So deadly was the contagion, so inadequate the methods of combating it, that even doctors and priests had to choose between death or flight. Worldly goods counted for nought.<sup>3</sup> Animals wandered about without an owner, goods lay open on all sides, and the harvest remained ungathered. The first shock over, the curious psychological phenomena associated with extreme and widespread tragedy showed themselves. Men tried to shut from their minds the monster of death and gave themselves over to idleness, selfishness and dissolute living. Fatalism became rampant and the Church, impotent in the presence of the disease, lost its hold. With the abandonment of mediaeval standards and restraints, society wandered in the wilderness. Contemporary writings refer to the laxity of individual and public morals, especially to the selfishness and avarice of the get-rich-quickly movement which set in in the late fourteenth century with the economic possibilities that were opening up.<sup>4</sup> The criticism of the Church is bitter and movements of succeeding centuries, such as the Reformation, merely added to the confusion of thought. The laxity in social standards was to continue until such time as

<sup>1</sup> See Henry of Knighton on this point.—No chaplain could be had under 10 marks or vicar under 20 marks.

<sup>2</sup> Political Poems I, p. 170. John of Bridlington.—Explanation of verses: "*Quinto ostendit auctor primum mysticum, scilicet primam magnam pestilentiam, factam anno Christi 1349, dicens. Cum canis intrabit, i cum illa still nociva in celo que canis primus dicitur oriatu cum sole, quod est quando sol est in fine cancri in mense Julii in diebus canicularibus qui sic dicuntur ab illa stella, tunc leo cum tauro volitabit i illa dua signa in celo, se quartili aspectu respicientia in celo circumvoluerunt et in leonem sol cito post intrabit et tunc ambo mordebunt canis et leo, per aeris pestilentiam quem cavebunt in terram mordebunt et destruent homines et letha manebunt i mortalitatem facient. . . .*"

<sup>3</sup> Henry of Knighton. "People cared little about wealth or things."

<sup>4</sup> Political Songs I, p. 279:—

*"Pax et patientia penitus orbantur;  
Amor et justitia domi non morantur;  
Errores et vitia gentes amplexantur;  
Patrum pro malitia parvuli necantur.*

*Heu! hunc mercenarii, nec veri postores,  
Rectores, vicarii mutaverunt mores;  
Ambitu denarii subeunt labores,  
Tales operarii merentur moerores."*

individual belief and individual responsibility could be evolved as the basis of human conduct in the modern individualist state. Herein lies the importance of Wicliffism and later of Puritanism.

## APPENDIX I.—ABERGAVENNY.

Ing, P. M., C. E., III. Files 91-92. Laurence de Hastings, Earl of Pembroke, and lord of Abergavenny. Inquisition at Werneurthrik (Abergavenny), April 17, 1349 . . . by the oath of 12 Jurors who say that the Earl held the manor of Penros from the king by knight service as parcel of the lordship of Bergavenny. There is there one messuage of no value beyond the charges; a fishpond without fish; 1 fulling mill formerly worth 33/4, now only 6/8 yearly because of the murrain; 142 acres  $\frac{1}{2}$  rood of arable worth 18/10 @ 1 $\frac{1}{2}$ d. an acre; 26 $\frac{1}{2}$  acres of pasture at 2d. an acre; (?) 27 acres of wood of no value because there are no buyers owing to the murrain. There used to be there of rents of assise of free and customary tenants £12 yearly but now only £4 and that because many of the tenements lie empty and derelict for lack of tenants. The works of customary tenants used to be worth per annum 22 0 $\frac{1}{4}$ , and now 16/- for the same reason. Pleas and perquisites are worth 13/4. Total £7 16 11.

Trefgaer Manor . . . There is 1 messuage worth nothing beyond the charges; 175 acres of arable worth 29/2 (@ 2d. each); 18 acres of pasture @ 2d. each—3/-. There used to be there of rents of assise of free and customary tenants £4 6 8, but now only 30/6 by reason of the mortality. Works of customers are worth 2/-. Pleas and perquisites of courts 5/- and no more owing to the same reason. Total 80/8.

Henllys Manor . . . 1 messuage of no value beyond the charges; 1 fishpond of no value because it is not stocked; 175 $\frac{1}{2}$  acs. of arable worth @ 2d. an ac., 29/2; 36 acres of pasture at 1 $\frac{1}{2}$ d.—4/6; 16 acs. of large wood with no underwood, and of no value; there used to be there of rents of assise of free and customary tenants 109 1 $\frac{1}{2}$  but now only 49/1 $\frac{1}{2}$  because of the mortality. Works of customers are worth 2/-. Pleas and perquisites of courts 16/- and no more for the same reason. Total 117/9 $\frac{1}{2}$ .

Trefgoythel. The Earl held in demesne as of fee a certain tenement at Trefgoythel . . . There is there 1 messuage of no value beyond repairs; 169 acs. of arable which are now worth nothing on account of the weakness of the land and the murrain (*morine*). The rents of assise used to be 70/6 $\frac{3}{4}$  but now only 6/- remain because of the mortality. There are no works of customers there and the pleas and perquisites of the courts are of no value because the tenants are dead. Total 6/-.

Bryngwyn. The manor and the advowson (40/-) held of the king in chief. There is one messuage of no value beyond the charges; there are 196 acs. of arable worth @ 2d. an ac. 32/8; 60 acres of pasture worth @ 2d. an acre 10/-; there are 111 acs. of wood of no value in underwood because of the murrain. There used to be there of rents of assise of the free and customary tenants 74/6 $\frac{1}{2}$  and now 28/2 $\frac{1}{2}$  because of the mortality. The works of the customers used to be worth 10/6 and now 3/-. The pleas and perquisites are worth 5/-. Total 78/10 $\frac{1}{2}$ .

Coyd Morgan . . . There is there one messuage of no value beyond the charges; there are 28 acs. of large wood of no value; three parts of one water-mill used to be worth 14/6 and now worth 3/- because of the mortality. There is another mill there now worth only 10/- for the same reason. There used to be of rents of assise £9 5 1 $\frac{1}{2}$  and now £4 owing to the same cause. There are no works of customers or pleas and perquisites. Total £4 13 0.

Werneryth. The Earl held in demesne as of fee at Werneryth . . . one park containing 80 acres of pasture worth yearly 10/- beyond the upkeep of the deer. There used to be of rents of assise £13 10 3 $\frac{1}{2}$  and now only 34/6 because of the mortality. The pleas and perquisites are worth nothing nor are the works of the customers. Total 39/1.

Llanwytherin . . . There is one messuage of no value beyond the charges. Two parts of a mill are worth 10/-; 20 acres of pasture are worth 3/4, there used to be of

rents of assise £4 4 0 but now 52/- owing to the mortality. Works of customers are worth 2/- and pleas and perquisites nothing. Total 70/7.

Llanover . . . The rents there used to be £12 but now £4 because of the said mortality. Three parts of 1 mill of Ogran which used to be worth 40/- is now worth 10/-. One fulling-mill which used to be worth 66/8 is now worth 13/4. Another mill which used to be worth 20/- is now worth 3/- because of the mortality. Total 6/8.

Ebbotfawr . . . There is a certain rent of £6 13 4 there and now only 53/4 remains because of the mortality; 12 acs. of large wood of no value. Total 53/4.

Ebbotfhechan . . . There are certain rents there, formerly £7 but now only 60/- because of the mortality; 10 acres of large wood there of no value. Total 60/-. Total value £34 2 3.

The above manors and lands were granted by Laurence de Hastings to William de Hastings, a bastard, who died March 9, 1349. At the time of the grant they were stated to be worth £120, but owing to the fall in their value a further enquiry was ordered (March 13, 26 E., III) for purposes of assignment. The enquiry of 1352 assessed the lands at £100 0 8½.

A special commission was then appointed to assess the entire lordship of Abergavenny both at the time of the death of the Earl (1349) and at the present date 1353.

#### APPENDIX II.

Ing. P. M., C. E., III. 96 (14). Lordship of Caus (Shropshire). (Robert de Harlegh.) (1349.)

Yokelton . . . "Two mills yield 10 small quarters of *tolcorn*—worth nothing owing to lack of grinding because there is no suit there and this because of the pestilence. Two carucates of land used to be worth 40/-, now worth only 6/8 owing to the pestilence. Rents of assise of free tenants—£8 but now only 30/- because of the pestilence."

Wentenore. Rents of assise of free tenants used to be £5, now only 40/- because of the pestilence.

Schelve. Rents of assise of free tenants used to be 36/-, now only 26/6 because of the pestilence.

Harlegh. In demesne are 2 carucates of land, usually worth 60/-; the jurors cannot extend the land because of the pestilence and no one wished to hire it. There are 3 acres of meadow, formerly worth 4/6, now 3/-, owing to the pestilence. Rents of assise of free tenants, formerly £4, now only 10/- owing to the pestilence.

Wyley. In demesne are 3 carucates of land but the jurors cannot extend the land owing to the pestilence. There are 4 acres of meadow, formerly worth 8/-, now only 4/- . Rents of assise of freemen, formerly 20/-, now 12/-.

#### APPENDIX III.—CHESTER AND FLINT.

Min. Acc. 783 1. Chester. (1354.) [of tenants.

<i>Old Castle.</i> —Decay of rents of 97 acres of new assart in the lord's hand for lack				
<i>Church Schocklach.</i> —	111	"	demesne	" 56/-
"	9	"	meadow	" "
<i>Pekforton.</i> —	44	"	demesne	40/6½
"	3½	"	meadow	" "
"	72 a. 1 rood	land	72/3	
"	135 a. 3 r.	demesne	77/4	
"	6 a. 1 r.	meadow	3/10	

Total £9 16 6

*Bunbury.*—In decay. 6/10 of the rent of 13/4 in decay for lack of tenants. 19/4 decay of 31 acres 3 r. " "

*Anderton.*—28 acres land in the lord's hands for lack of tenants, 11/4

*Hurleston.*—5½ " " " " 2/9

*Brundelegh.*—33 4 from food of the "satellites" (i.e., serjeants).

Min. Acc. 788/15. Chester. (1350.)

A/c of Bailiff of Middlewich. Rent called *Kynggesmol* respited owing to lack of tenants, 4/9.

A/c of the Escheator. Wenere and Mondrem.—“Of the 8/- charged for 1 messuage and 1 bovat in Russheton which formerly belonged to Thomas de Wenere. Nothing, because the messuage was not occupied owing to the pestilence, and the bovat lies in stable and uncultivated.”

Chelmondeston.—4/4 issues of 1 *place* of land in the lord's hand 19 E. III.

Now the *place* lies barren because of the pestilence and is common.

Kengeslegh.—Of 20/-, *relief* for the manor of Aston. Nothing; no inquisition could be taken because of the pestilence.

[Note.—Numerous deaths are recorded and payments of relief.]

Min. Accs. 788/16; 788/17. Manor of Frodesham. 1351 and 1352.

1351—Rents of messuages, bovates, &c., in decay, total 61/-.

1352—       “       “       “       “       81/9.

Min. Acc. 1186/4. Hopedale. (1350.)

Park of Llwydcoed. 25/10 from agistments of the park. No more because few animals were agisted there this year because of the pestilence in the previous year.

County of Flint. 4/- from the miners of lead of Englefield. No more because the miners there are dead for the most part and those who survived are unwilling to work there.

Min. Acc. 1186/5. Town of Rhuddlan. (1351.)

Allowed to the Burgesses £13 6 8 remitted by the lord, part of the farm of £40 for the mills of Disserth and Pentref, for this year and so from year to year until the said mills are of more value. Allowed £10 of the farm of the said mills for last year because the farm could not be levied because of the poverty of the people owing to the pestilence.

County of Flint. Farm of Advowry—30/- with farm of the *Rhaglawry* and other offices—couldn't be let because of the pestilence. 13/- debts for fines and amercements at the Sessions and other debts—could not be levied because of the pestilence.

The Min. Accs. for Flint, 1186/11 (1355); 1186/23 (1362); 1187/11 (1369); 1187/12 (1370), especially the last, contain long lists of lands escheated and alienated (often without licence) including “New Escheats,” also “Native” land in the lord's hands. No direct reference is made to the Pestilence but the successive epidemics in Flint would appear to have been very severe.

#### APPENDIX IV.

Min. Acc. 1182/3. Lordship of Denbigh (28-32 E. III.)

Uwchalet Commote—(from Easter 1354 for 22 weeks). Allowed 54/1 from the customs of divers tenants of the *villatae* of Barrok and Petruall in the lord's hands because the tenants died in the time of the pestilence and their heirs are unable to take up their inheritance.

Uwchalet Commote (Michaelmas 1354 to Michaelmas 1355). Allowed *tunk* (commuted food rents) and custom of butter from 2 *gavels* of land in Llechtalhaegan in the lord's hands for lack of service of the tenants, 20/10. Loss of ‘*tak*’ and custom of divers tenants of the *villatae* of Barrok and Petruall this year, £4 4 1. Total £5 4 11.

Uwchalet Commote (1355-1356). Allowed for the two *gavels* (as above) 20/10. (Also for years 1357 and 1358.) Respited, divers *reliefs* amounting to £16 15 8.

Lordship of Denbigh. Escheators' accounts. These accounts reveal very great changes in the lordship during the years 1350 to 1370, as the result of the escheat and transfer of lands both in the manors and in the *vills* of the Welsh. Heavy losses in



rents are recorded, the arrears being recorded on the Great Roll of Debt of the Lordship. The details hardly lend themselves to statistical treatment, but the following extracts may indicate some of the immediate effects of the waves of epidemic, e.g.

Min. Acc. 1233/2 (1355). "Issues from sales of chattels and from the produce or rents of escheated lands, viz.:

In the Englishry and in Caemerch Commote	...	£17	17	5½
In Uwchalet Commote	...	12	7	7½
" Isdulas	...	9	16	4½
" Uwchdulass	...	23	2	1
Total	...	£75	14	8

[14/- issues of 38 acres of land . . . put in herbage this year for the tenants of the villata of Dynorbyn Fychan (Isdulass), and no more because the tenants of the vill and other vills adjoining are *nativi* of the lord and poor that they can scarcely keep up their own land.]"

Min. Acc. 1182/4 (1361).

Segroid. Nothing from lands in decay in the park of Segroid owing to lack of buyers.

Kermyrenet. From the 3/- for herbage—nothing, because the tenants are so poor that they can scarcely keep up their own land. [Similarly in Sceibion.]

Issues of the Welsh Lands:—

Prees. 14/8 issues of divers Welsh lands in the lord's hands and let, beyond the services.

Llechryd. 1/-.

Nanthyn Canon. 5/10, &c., numerous lands in the lord's hands owing to failure to pay relief.

Commote of Isdulass. 10/- for issues of 40 acres of land of the vill of Dynorbyn Vawr which used to render 19/8, let in grass this year. No more because no tenants live in the same vill. There are now no mills at Kylkeyn, Bodelennon, Massegwyk, and Glyngewy.

Abergele. 48/8, issues of lands let, &c., &c.

Total of issues from each commote: Uwchdulass, £6 14 7½; Isdulass, £4 19 7; Caemerch £2 11 6½; Uwchalet £2 0 11; Isalet £14 9 2½.

[The debts of the ministers for their several bailiwicks are also very heavy.]

Min. Acc. 1182/5 (1362). Denbigh Lordship.

Total of rents respited £46 11 4½. Losses in rents for lands farmed and for vacant tenements.

	Caemerch	Isalet	Uwchdulass
(Long list)	£8 13 4½	£11 11 0½	5 3 6
		3 7 4½	1 15 11
		6 6 8 (vacant bovates, &c.)	

Min. Acc. 1183/2 (1370). Denbigh Lordship.

Englishry. Payments of relief respited, £7 10 6½.

Caemerch Commote. Loss of customs and services of 7 *nativi* @ 1/2 a year each = 8/2. [Similarly for the previous two years.] There are now only 24 tenants instead of the previous 31. Loss of services of 9 freemen and free tenants = 1/1½. [Similarly for the previous two years.] There are now only 36 tenants instead of the previous 45. Decay of services of 1½ part of a *gavel*, 7½d.

Commote of Isalet. Loss of customs and services of 3 *nativi* for the years 1365 and 1364, 19/1½. [There were only 45 *nativi* instead of 48.] By 1373 the 48 *nativi* had been reduced to 35. Min. Acc. 1183/9. Loss of services, &c., of 8 *nativi* lacking this year, 28/-. Total of rents, *reliefs*, &c., respited, £16 15 0½.

Commote Uwchdulass. Rents, *reliefs*, &c., respited, £7 12 3.



Dynmael. Rents and services of various *gavels* and other rents in decay  
£4 17 5½.

Commote Caemerch.—

Gavel Rhydd.	Jeuau ap Madoc, 10d.	} Nothing; they are dead.
	Howel Goch, 2d.	
Carwethynydd.	Issues of the rights of Dd. ap Teg-let	} Nothing; they are dead.
	" " Howel Goch (1/4)	
	" " Jeanu Goch, 2/-	
Prees.	Issues of 35 acres (17/6), 10/-.	} Nothing; they are dead.
	" Rights of Jenau ap Jorwerth and another, (1/4) 1/-.	

Min. Acc. 1183/3. Receiver's Account for the Lordship of Denbigh (1370).

Hanassok, 8d. *recognition* of Ithel Agan, *nativus* of the lord, because he died and his heir holds 'native' land in Landrol.

Commote of Caemerch. Decay of rents farmed, lands, pastures, &c., in the lord's hand for lack of tenants, £20 13 5½.

Commote of Isalet.	Defect of rents, &c.,	£19 2 3½	}
	Decrease of rents farmed,	£4 2 2½	
	Rents allowed for bovat lands, £2 6 8		

Commote of Uwchdulas.	Decay of rents farmed, 102 5	}
	Decrease of rents, 46 7	

[Cf. also Min. Accs. 1183/4-13.]

Min. Acc. 1233/5. Escheator's Account. Lordship of Denbigh. (1373.)

Total of issues of escheated lands together with arrears = £232 10 2.

#### APPENDIX V.—(A) ANGLESEY.

Min. Acc. 1149/1. Anglesey. (1351.)

M. 3. Commote Malltraeth.

"Issues of the Manor—18/2 received from the herbage of divers old stubble lands of the *nativi*, in the lord's hands for lack of tenants in the vill of Dystenydd . . . &c. Total 61/11.

New decays. Decay of rents of David Moil of the vill of Trefdisteinydd 5/4. Gronou With 3/4. Turlagh 22/4. Madoc ap Jack 7/- and Jeuau ap Gronou of the same vill 10/10; Cad. ap Jak of Trefithon 5/4 and 7 others of the same vill; Howel ap Teg of Dyndroval 5/-; Hova Goch of Rhosmawr 10d., and 18 others named in the same vill which lands are 'native' and in the lord's hand for lack of tenants and because now the lord's tenants in the same vill are unable and insufficient (*impotentes et insufficientes*) to hold the said lands." Total of the new decay £6 16 2.

Commote Llifon.

Issues of the Manor. Total 52/11.

New Decays of Land of the *Nativi*. In decay of rent of the land of Trefdolfyn of lands of the *Nativi* in the lord's hand for lack of tenants—20/10 [of which 10/10 for rents of assise, customs and services in the same vill and 10/- from the same *vill*, part of 42/7½ from all the villeins of this commote] and of land of Trefcyclok (al. Trefedol) 6/8.

" " Gronou ap Eynon, 6/8.

" " Mab Rirert, 3/4.

" " Howel ap David, 9/6.

" " Trefmeddygon (the doctors' tref) (in Tref Meibion Meuria) 6/-.

" " Adam Gutta and 3 others, in Bodenawlwyn, 44/6.

" " Llew. ap Meredith 8d. and Llew. Benhir 8d. which lands are of the lord's villains and in the lord's hand for lack of tenants and because the *nativi* surviving in the same commote are unable (*impotentes*) to hold the said lands. Total of New Decays—£4 18 10.

Commote of Talybolion.

Issues from Escheated Land.—6/2 from herbage of the land which was of Meredith Benhir in the lord's hand in Llanbugel . . . &c. (5 other entries).

Issues from *Native Land* (in lord's hands).—14/6, herbage of villain land in Aberalaw which Madoc ap Philip and 4 others, *nativi*, held for 32/2, in the lord's hands, because the *nativi* who survived after the pestilence were insufficient on account of their inability to hold the lands.

12/7, herbage of land of Map Eynon and 5 others in Carnedawr (held for 22/3).

5/- " " Map Gwyon in Meryogen (10/6).

17/6 " " native-land of 4 *nativi* (named) in Llannol (30/-).

5/9 " " 2 " Cotrounyn and 2 in Kasvan (2/6).

Total of Issues—£4 11 10.

Lands in decay.—Decay of rent of land of Meredith Benhir in Llanbugel because none could hold the tenement, 15/6.

" " ½ part of the mill there, 1/-.

" " 30 acres of Llewelyn ap Meredith there, 26/8.

" " land—Jorwerth ap Madoc in Trefwadok, 3/4.

" " 20 acs. of land in Cardecank, 8/0½.

" " 1 messuage, 10 acres land, ½ ac. of mead . . . of Eden ap Gronow, 11/2.

" " 5 bovates of land and easements of Traharn ap Blethin, in Canternok.

" " Native Land because the *nativi* were unable and insufficient owing to their weakness and poverty to hold the land—107/5, viz., for the land of Madoc ap Philip, &c. 10/8, &c., as in the 'Issues' above, viz., 19 entries for the entire commote [viz., in Aberalaw, Carnedawr, Meryogen, Llannol Cottrounyn].

Total of rents in decay £9 8 3½.

Respited of the £26 1 4½—£6.

Commote of Tureclyn.

Allowed £6 9 11½, viz., 6/5 (part of 11/10) rents of the villains of Curthlayet in the lord's hands for lack of tenants . . .

53/4 (part of £6 17 4) rents of the villains of Cotunot . . . because the 5 villains surviving are unable to hold the vill and could scarce pay £4 0 2.

11/6 (part of 52/1½) rents of the villains of Rodevyney because there are only 2 tenants there (1 *nativus* and 1 *advowry*) rendering 10/2 and 1 tenant called Deyok (who used to render 17/4).

1/8½ (part of 3/4½) for 1 messuage 3 bovates of land which were of David Rurith, escheat for lack of tenants.

1/4 (part of 1/10) for escheat on failure of heirs in the lord's hands for lack of tenants. Total of allowances—£6 9 11½.

Respited. 16/5 rents of free tenants for this year and last in the lord's hands. No heirs claimed.

Lands of freemen in the lord's hands for 1350—15 entries. Also 3 other entries (5 persons).

Sum of rents of freemen for year 1350—16/8, and for the year 1351—4/10.

Min. Acc. 1149/9. Beaumaris Borough. (1360.)

Respited 9/6, rents of 9½ burgages because the burgages stand empty and uninhabited for lack of tenants.

[See Extent of Anglesey 1294 for a list of the vills and their rents.]

#### (B) CARNARVON.

Min. Acc. 1171. County of Carnarvon. (1351.)

Commote of Creuddyn. (1351.)

8d. Advowry of Blethin ap Madoc ap Robyn and David ap Madoc and no more because the remainder are dead by Pestilence. (Formerly the receipts were

3/- i.e., 9 advowry tenants.) From the reliefs of *advowry* tenants dead whence the lord has from each, one-half of a mark and from the goods of the same which the lord shall have if he died without an heir.—Nothing, because none died (i.e., during this year).

Farm of the Office of *Rhaglaw* which used to be 43/4 before the pestilence,—Nothing, because it was let to Madoo Blodeydd for 26/8.

26/8—farm of the offices of *Rhingyll* and Woodward let. And they used to be let before the time of the pestilence for 20/- and 13/4 respectively.

5/4 from half of the value of 2 cows which the community of the commote used to give to the lord's lardar for munitioning the castle of Conway. A cow is appraised at 5/4 this year. The half of the value of one ox which the lord was accustomed to have from the community for the same store is not received because it was remitted owing to the poverty and the fewness of the tenants.

Commote of Arllechwedd Isaf.

Arachlehan. 40/- annual rent of 19 bovates of land passed into oblivion on the making of the extent which vill E. ap Gruffydd, deceased, held at the King's will.

5/2 rent of customs and services of the King's villains of Morva Vychan for 13 bovates of land and easements which the same villains held and which passed into oblivion on the making of the extent.

10/- from the entire commote for the upkeep of the buildings, which is called *Têth*, in the manor of Aber, which upkeep was committed to oblivion on the making of the extent.

3/- from *Advowry* and no more because many are dead by the pestilence.

40/-, farm of the *Rhingildry* and before the pestilence it was 71/6.

6/8 from half of 2 oxen and 2 cows which the community of the commote give to the larder of the lord for munitioning the castle of Conway beyond the other half of the same store which the lord used to pay. And no more because of the poverty and fewness of the *nativi* and *advowry* in the commote.

Allowed 8/5 rents of various lands in the lord's hands for lack of tenants.

Commote of Arllechwedd Uchaf.

No *advowry* tenants died.

14/-, half of the value of 3 oxen and 3 cows . . . from *nativi* and *advowry* of the commote. No more because of the paucity and poverty of *nativi* and *advowry*.

Commote of Nant Conway.

3/4 from *Advowry* tenants; and no more because they are dead by the pestilence.

14/- from the community for the lardar; and no more owing to the cause above. Respited. 5/8 rents of divers freemen whose lands and tenements were in the lord's hands because no heirs claimed.

Commote of Iscorvey.

13/4 from the community for the store of the Prince. And no more because of poverty, etc.

Respited. 22/- for this year and last year, rents of lands in the lord's hand.

Commote of Uwchcorvey.

14/- from the community for 2 oxen and 2 cows for the store of the Prince and no more, owing to the poverty and paucity of the tenants.

Respited. 8/4 rents of lands in the lord's hand.

Min. Acc. 1171/8. Carnarvon. 1362.

Commote of Creuddyn.

Deganwy. 3/5½ from 3 Welsh acres, formerly of D. ap David, *nativus* of the king of the same *vill* which came into the hand of the Prince . . . in the 1st year of the principate by the weakness (*impotentia*) of the said *nativus*.

For the Store of the Prince (*Staurum principis*), viz., 1 ox and two cows from the customary tenants and the *advowry* and if there are not enough of the Community of the Commote, as mention is made in the Charter of Lincoln made to the community of Wales concerning the store of the castle of Conway, beyond the other half of the value which the lord is responsible, they will answer nothing this year because the lord commanded by letters to the Justice . . . that he should make respite to the said villains and *men of advowry* for this store from the time of the pestilence until it should please him, &c. . . .

(Letter dated July 30, 26 E. III.)

Respited. 65/7 of which 21/3½ part of 41/3½ were for divers rents and services of villein lands in Gannowe Vill (Deganwy) because the land remained in the lord's hand for lack of tenants and are let to Glodyth for a term of 8 years (this year the second).

Commote of Arllechwedd Isaf.

1/4 from 2 bovates of land which were in the hand of the vill of Llaneanon and committed to oblivion but now let at farm.

Drammis. 8/2½ from 13 bovates of land with easements, pasture and wood, formerly of Jeuan ap Philip and Madoc his brother, *nativi* of the king in the *vill* of Llegham, which came into the king's hands because the said villains died without heirs of their body.

6d. from 2 bovates of Heilim ap David and Madoc ap David, brothers, *nativi* . . . now handed over to oblivion.

40/- rent from 19 bovates of land in the vill of Driannus, passed into oblivion and Eden ap Gruffydd lately held it at will, etc., etc.

Dynllaen. Store of the Prince, 6/8 only.

Trefgarnedd. 12d. from the *vill* of Llediok which is in villeinage and in the hands of the king for lack of tenants let beyond the services and rents for 4 years.

Manor of Hirdref. £4 6 8 farm of the manor let to Gruffydd, which *vill* used to answer for 66/- beyond the issues and is extended yearly at 114/-, viz., 6/- for 2 bovates of land and meadow; 2/- for ½ acre of pasture; 36/- rent of 19 villeins; 54/- from the same for 36 crannogs of oatmeal, 8/4 from the same for works in autumn and winter; 6/- from the same for farm of the mill and 1/4 for the works of the mill. But it could not be assessed beyond this year because many of the tenants are dead by the pestilence.

Boidellas. 7½d. from 2 acres of Welsh land which were of the 3 sons of Ithel Coeh . . . which are sown and which are in the king's hands owing to the impotence of the same.

Released from £21 11 0 for the rents of divers lands and tenements in divers *vills* in this commote for this year and year 25, granted to Thomas de Brerele, Kt. and Ida his wife with the manor of Bodevan and other lands in the commote of Llifon in the County of Anglesey to be held for £50.

Commote of Cafflogion.

24/11 from lands and tenements of free tenants in the lord's hands owing to impotence.

Released from £8 2 2, viz., £4 2 2 for this year and the last two years for the lands of divers freemen lying empty and uncultivated because there are no heirs.

Min. Acc. 1171/9. Carnarvonshire. (1363.)

Commote of Cafflogion.

24/11 from lands in the Prince's hands, 6 bovates in Bodenith and Carigoll, 6 bovates in Penlegh Vachan in villeinage in the lord's hands for lack of tenants.

Respited. £4 8 11 for lands in the commote in the lord's hands owing to failure of heirs.

Commote of Eifionydd.

In decay of the rents of the vill of Redemknelyn which was in the hands of the villains but vacant since the pestilence and extended at 28/- for 8 *crannogs* of oatmeal and 3 *dishes* of butter whence charged above 5/- and no more because 23/- were raised from the pasture of the *vill*.

Respited. 28/1½ part of 37/3½, rents of land and tenements of free tenants in the lord's hands because there were no heirs and nothing could be raised from the lands.

Remitted to the tenants of the lord, *nativi* and *advowry* in this commote £19 11 8.

#### APPENDIX VI.—MONTGOMERY.

Min. Acc. 1206/3. Montgomery (1365).

Town of Montgomery. Respited £6 13 4 (part of 20 marks) of the farm of Dd. ap Owen.

Respited £1 9 7 for loss of rents of divers lands and tenements in the town of Montgomery for this year and for the last 4 years (i.e. since 1361) viz., @ 5/11 a year, because the tenements are vacant and the tenants are not in the patria.

Teirdref. 3/9 for 'cylch' of the tenants resident in the lordship each giving 1d. at Christmas for ancient custom. No more because there were no more tenants resident there.

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NO other chemist of Islām can compare with Jābir ibn Ḥayyān in celebrity. Yet up to the present time there has been no attempt to gather together the information concerning him which is scattered through Muḥammadan chemical literature. The vexed question of the possible identity of Jābir with the famous author, "Geber," of the *Summa perfectionis* and other works well known in a Latin form—a question which has provoked much controversy during the last fifty years—is reserved for discussion at a later date. It is obvious that a comprehensive study of the works of Jābir ibn Ḥayyān is an essential prerequisite. The conclusion of the pseudepigraphists, namely, that Jābir is not identical with Geber, may be correct, but it is impossible to deny that the evidence upon which this conclusion has hitherto been based is inadequate, often untrustworthy and not seldom absolutely incorrect. While, therefore, this article is primarily intended to be a contribution to our knowledge of the authentic and historical Jābir, it may also throw light indirectly upon the "Geber" tradition.

### (1) BIRTHPLACE AND LIFE.

Al-Nadīm<sup>1</sup> says that Jābir's full name was Abū 'Abdullah Jābir ibn Ḥayyān ibn 'Abdullah al-Kūfī. He is, however, almost always called elsewhere Abū Mūsā, not Abū 'Abdullah; he may have had two sons, Mūsā and 'Abdullah, in which case either name would be correct. Al-Qiftī states in his *History of the Sages*<sup>2</sup> that Jābir excelled in the natural sciences, especially that of chemistry, and wrote numerous well-known books. He was, in addition, skilled in philosophy and esoterics, and was a Ṣūfī. Ibn Khallikān<sup>3</sup> describes Jābir as a pupil of the Imām Ja'far al-Ṣādiq (699-765 A.D.), a tradition which is mentioned also in the *Fihrist* and in certain of Jābir's own writings. Ḥajjī Khalifa's statement that Jābir was a pupil of Khālīd ibn Yazīd ibn Mu'awiya (died 704 A.D.) need not be taken literally, and probably means simply that Jābir was a student of Khālīd's books: it is, of course, well-known

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that Khalid was the earliest Muslim of rank to take an interest in chemistry, which he is said to have learnt from the monk Marianus.

The Fihrist gives the titles of several books which Jābir wrote for the Barmekides, and in his *Great Book of Properties* Jābir<sup>1</sup> refers by name to Khalid, Ja'far and Yahyā, members of the Barmekide family. He seems, indeed, to have been on terms of some intimacy with them, as we shall see later.<sup>2</sup> The Barmekides enjoyed the favour of Hārūn al-Rashid for seventeen years—from 786 to 803 A.D.—so that we can safely place Jābir's youth prior to 765, the date of the death of Ja'far al-Sādiq, and his manhood in the last quarter of the eighth century. The exact date of his birth is unknown, as is that of his death. According to Hājji Khalifa<sup>3</sup> he died in A.H. 160, that is 776/777 A.D., but this date is obviously incorrect, from the above considerations. Aidamir al-Jildaki (died about 1360 A.D.), who was extraordinarily well-informed about the chemists of Islām, states<sup>4</sup> that Jābir became involved in the fall of the Barmekides in 803 A.D. and had to fly for his life to Kūfa, where he lived in retirement till the days of the Caliph al-Ma'mūn, who succeeded to the throne in 813.

From these facts it would appear reasonable to place Jābir's birth somewhere about the years 730-735 and his death some eighty years later; it must, however, be definitely stated that these dates are only conjectural. All that we can be certain of is that he flourished during the latter half of the eighth century, and that the date given by Hājji Khalifa for his death is much too early.<sup>5</sup>

Jābir is variously described as "of Tūs,"<sup>6</sup> "of Tartūs,"<sup>7</sup> "of Kūfa,"<sup>8</sup> "of Khorāsān,"<sup>9</sup> and also as a Sabæan (of Harrān).<sup>10</sup> No information as to his birthplace is to be found in his writings, but there are traces of Sabæan doctrine in some passages; it would, however, be unsafe to assume that his belief in the influence of the stars necessarily implies that he was a Sabæan, since this belief was common to all his contemporaries. Practically all the authorities agree in stating that he lived at Kūfa for at least part of his life. The Fihrist<sup>11</sup> relates that, over a century after Jābir must have died, while some houses in the quarter of Kūfa known as the Bāb al-Sha'm were being demolished a mortar containing over 200 lb. of gold was found on the site of Jābir's laboratory.

Since the Barmekides were in constant attendance at the Court at Baghdād, it may be assumed with reason that for some years Jābir lived in the capital of the empire. Of his life there we know very little, but he paints one or two sketches, in the *Great Book of Properties*,<sup>12</sup> which show us that he practised

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<sup>9</sup> *Fihrist*, p. 355.

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medicine as well as chemistry. Thus he says<sup>1</sup> that he cured a valuable slave-girl, belonging to Yahyā ibn Khālid, by means of an elixir he had prepared, and that Yahyā was so impressed by the cure that he began to study science "and persevered until he knew many things; but his son, Ja'far, was cleverer than he."

He seems to have suffered persecution at times. The Fihrist says that he did not long dwell in one spot, as he feared for his life, and Al-Jildakī<sup>2</sup> affirms that "Jābir ibn Ḥayyān narrowly escaped death on many occasions, and met with affliction and violence on account of this science [i.e. chemistry] from the hands of envious and evilly-disposed people. He finally had to explain some of the science openly to Al-Rashid and to Yahyā ibn Barmak and his two sons Al-Faḍl and Ja'far, whence their riches."

## (2) WORKS.

Jābir was a voluminous writer. His own "Fihrist" or list of his writings, which was used by Al-Nadīm,<sup>3</sup> is unfortunately now lost. Al-Nadīm's list is incomplete, and in one case at least an error has been made by Fluegel and his collaborators in editing the titles given in Al-Nadīm's *Kitāb al-Fihrist*.<sup>4</sup> Berthelot's translation<sup>5</sup> of the titles given in the *K. al-Fihrist* is often inaccurate and unintelligent. The majority of the books of Jābir here mentioned have been lost, so that no useful purpose would be served by reproducing the list in its entirety. We shall, however, give the names of (a) those books mentioned in the Fihrist which are still extant, either complete or in fragments; (b) those which have some interest for other reasons, and (c) those which are not mentioned in the *K. al-Fihrist*, but which are known in MS. or printed, or known by title only.

### (A) BOOKS MENTIONED IN THE FHRIST OF WHICH MSS. OR PRINTED EDITIONS ARE EXTANT.

(1) *Kitāb Istuqūs al-'Uss al-Awwal*. Lithographed. India, 1891. Berthelot (op. cit.) naively translates this title "*Le Livre d'Estaques, le premier myrte*," thus confessing ignorance of the meaning of *istuqūs* and misreading *ūs* (myrtle) for *'uss* (base or foundation). *Istuqūs* seems to be the Greek *ἰστυκός*, which is used by Aristotle (*Metam.*) in the sense of firm, solid, or substantial. Here it is used in the sense of "foundation," so that the title may be translated "*The Book of Foundation, the First Base*." That this is the correct rendering is shown by Jābir's own explanation in his book (No. 4) *Tafsīr Kitāb al-Istuqūs* ("Commentary on the Book of Foundation") where he says that he has placed the Book of Foundation in three parts [of which the present is the first], at the beginning of his "112 Books," because it contains "the sum of the meanings" of the rest, and so serves as an introduction or prolegomena to them. "To whomsoever understands it is manifest all that which is in the 112 Books." "*Istuqūs*," he says, "in the Greek language signifies the foundation of a building."

Salmoné<sup>6</sup> gives the word as *istaqs*, meaning "the four elements," while Dozy<sup>7</sup> gives *istūqīs*, quoting Freytag, and *ustuqussu* on the authority of Schiaparelli.

<sup>1</sup> Op. cit., *maqāla* 6.

<sup>2</sup> *Nihāyat at-Talab*, ii, end.

<sup>3</sup> *Kitāb al-Fihrist*, ed. Fluegel.

<sup>4</sup> *Vide infra*, pp. 50, 51.

<sup>5</sup> That is, the translation published by Berthelot, op. cit., iii, 32-36. The translation was actually made by O. Houdas.

<sup>6</sup> Arabic-English Dictionary, London, 1890, p. 13.

<sup>7</sup> *Suppl. aux dictionnaires arabes*, Leyden, 1881.

(2) *K. Istuqus al-'Uss al-Thānī*. Lithographed. India, 1891. The second part of the same work.

(3) *K. Istuqus al-'Uss al-Thālith*. Lithographed. India, 1891. The third part of the above, apparently that given in the *Fihrist* as (No. 43) *K. Istuqus* merely.

(4) To the above should be added the *Kitāb Tafṣīr al-Istuqus*. Lithographed. India, 1891. "An Explanation of the *Istuqus*." Not mentioned in the *Fihrist*.

(5) *K. al-Wāhid al-Auwal*. "The First Book of Unity." *Bibl. Nat. Arabe* 2606. Apparently the same as the *K. al-Wāhid al-Kabīr* in the *Fihrist*.

(6) *K. al-Wāhid al-Thānī*. "The Second Book of Unity." *Bibl. Nat. Arabe* 2606. Apparently the same as the *K. al-Wāhid al-Saghīr* in the *Fihrist*.

(7) *K. al-Rukn*. "The Book of the Fundamental Principle." This is probably the same as the *K. al-Arkān* [*arkān* is the plural of *rukn*], from which a short quotation is made in section 7 of the *Rutbatu'l-Hakim* ascribed incorrectly to Maslama al-Majritī. A *K. al-Arkān al-Arba'a* is mentioned by Jābir in his *K. Nar al-Hajar*, q.v.

(8) *K. al-Bayān*. "The Book of Explanation." Lithog. India, 1891.

(9) *K. al-Nūr*. "The Book of Light." Lithog. India, 1891. Steinschneider (*Die europ. Uebersetz. a. d. Arabischen*, A, 73; Wien, 1904), speaking of Geber's *Lib. Fornacum*, says "Unter Djābir b. Hajjans Schriften im *Fihrist*, S. 355, kommt nur Z. 29 'Kitāb al-Nur' vor, welches aus Tannur entstanden sein könnte." However, the *K. al-Nūr* is an entirely different work from the *Lib. Fornacum*. Incidentally it may be mentioned that the translator of the *Lib. Fornacum*, given in *Artis chem. principes* (Basle, 1572) as Rodogerus Hispalensis (see Kopp, *Beiträge z. Gesch. der Chemie*, III, 34—not 84 as quoted by Steinschneider), is called 'Roger Bacon Hispalensis' in B. M. Sloane 1118 fol. 60—a fifteenth century MS. 'Roger Bacon' here is apparently a misreading for Rodogerus. Who Rodogerus was I have not been able to discover.

(10) *K. al-Zaībaq*. "The Book of Mercury." Berthelot (op. cit., iii) prints two "Books of Mercury"—*K. al-Zaībaq al-Sharqī* (Book of Eastern Mercury) and *K. al-Z. al-Gharbī* (Book of Western Mercury) from the Leyden MS. *Arabe* 440. They are also in *Bibl. Nat. Arabe* 2606.

(11) *K. al-Sha'ar*. "The Book of Hair." *Brit. Mus. Addl.* 7722, No. 5.

(12) *K. al-Tabwīb*. "The Book of Arrangement by Chapters." This is the *Bibl. Nat. MS. Arabe* 2606. It is also quoted by Al-Tughhrā'ī, *Brit. Mus. MS. Or.* 8229.

(13) *K. al-Durratu'l-Maknūna*. "The Book of the Guarded Pearl." A MS. of this title, anonymous, is found among works of Jābir in the British Museum. *MS. Addl.* 7722, No. 11.

(14 and 15) *K. al-Shams*. "The Book of the Sun, or Gold," and *K. al-Qamar*, "The Book of the Moon, or Silver," are probably extracted from the *K. al-Ajsūd al-Sab'a*, "The Book of the Seven Metals," quoted several times by Al-Jildakī in his *Nihāyat at-Talab*. See also *Bibl. Nationale MS. Arabe* 2606, and Nos. 71 and 72, below.

(16) *K. al-Tarākīb*. "The Book of Combinations." *Bibl. Nat. Arabe* 2606. This possibly is the *K. al-Tarkīb* of the *Fihrist*.

(17) *K. al-Haiyawān*. "The Book of Animals." A book of Jābir's, entitled *K. Hayyatu'l-Haiyawān*, "The Book of the Life of Animals," is quoted by Al-Jildakī (op. cit., vol. i).

(18) *K. al-Asrār*. "The Book of Secrets." This may be the same as the *K. sirr al-Asrār*, "The Book of the Secret of Secrets," of which there is a MS. in the *Brit. Mus. (Addl.* 23418, No. 14) and which is mentioned with quotations several times by Al-Tughhrā'ī (*Brit. Mus. Or.* 8229). There is a Latin MS. *Secreta Secretorum* ascribed

to "Geber" in Gonville and Caius College, No. 181, and in Corpus Christi College, Cambridge, No. 99.

(19) *K. al-Ard*. "The Book of the Earth." A work of Jābir, *K. ard al-Hajar*, "The Book of the Earth of the Stone," is printed by Berthelot (op. cit., iii) from the Leyden MS. *Arabe* 440. It is also in Bibl. Nat. *Arabe* 2606.

(20) *K. al-Tarkīb al Thānī*. "The Second Book of Combination." Bibl. Nat. *Arabe* 2606.

(21) *K. al-Khawāss*. "The Book of Properties." Brit. Mus. Or. 4041 and *Addl* 23419 No. 2.

(22) *K. al-Tadhkīr*. "The Book of Admonition," or "The Book of Rendering Masculine." There is an anonymous MS. of this title, occurring among works of Jābir, in the Brit. Mus. (*Addl* 7722, No. 12).

(23) *K. al-Istilmām*. "The Book of Demand (or Search) for Perfection." A few quotations are made from this book by Al-Tughrā'i (Brit. Mus. Or. 8229), and also by Al-Jildakī in his *Nihāyat al-Talab*. The title corresponds to that of the *Liber de Investigatione Perfectionis* of "Geber."

(24) *K. al-Aḥjār*. "The Book of Stones." A *K. al-Hajar* ("Book of the Stone"), of Jābir, was lithographed in India in 1891.

(25) *K. al-Rauḍa*. "The Book of the Garden." Quoted by Al-Jildakī in vol. ii of his *Nihāyat al-Talab*.

(26) *K. al-Munāfi'*. "The Book of Advantages." A book by Jābir entitled *K. Munāfi' al-Hajar*, "The Book of Advantages of the Stone," is in the Berlin MS. 4199, i.

(27) *K. al-Idāh*. "The Book of Explanation." Lithographed. India, 1891.

(28) *K. Musahhiḥāt Flātūn*. "The Book of Emendations of Plato." MS. at Constantinople (Defteri Kutubhāne'i Rāgib Pāshā, 96, No. 4, Stambul, 1310 A.H.).

(29) *K. al-Damīr*. "The Book of Secret Thoughts [or, 'of the Pronoun']." Bibl. Nat. *Arabe* 2606. This is mentioned by Al-Jildakī, op. cit., vol. ii, end, under the title *K. al-Damīr fī Khawāss al-Iksīr*, "The Book of Secret Thoughts on the Properties of the Elixir."

(30) *K. al-Mawāzīn*. "The Book of Balances." Printed by Berthelot (op. cit., iii, p. 105 of Arabic text) from the Leyden MS. *Arabe* 440. I presume this work is the *Liber de Ponderibus artis*, Borellius, *Bibl. Chim.*, Paris, 1654, p. 103.

(31) *Kutub al-Mulk*. "The Books of Dominion [or, 'of the Kingdom']." In the Fihrist, Jābir says, "I composed a book known as the Books of the Kingdom." This appears to indicate that the book referred to consisted of two or more smaller books included under one title, and this is borne out by the following facts: Berthelot (op. cit., iii) printed the text of a *K. al-Mulk* from the Leyden MS. *Arabe* 440; there is another copy of the same work in Bibl. Nat. *Arabe* 2605. But this *K. al-Mulk* is quite different from another of the same title lithographed in India in 1891.

A *K. al-Mulk* of Jābir seems to have been translated into Latin, as a *Lib. Regni* of 'Geber' is mentioned by Borellius (*Bibl. Chim.*, Paris, 1654, p. 103), and by Carini, *Rivista Sicula*, vii, pp. 175 and 179.

(32) *K. al-Riyāḍ*. "The Book of Gardens." Bodl. Marsh 70, Brit. Mus. *Addl* 7722, No. 5.

#### (B) BOOKS OF INTEREST, UNKNOWN IN ARABIC, OR REQUIRING SPECIAL MENTION.

(33) In the list given in the Fihrist occurs the title *Kitāb ilā Qalamūn*. Berthelot translates this as "Livre à Qalamoc; peut-être faut-il lire 'Le livre du Caméléon.'" Fluegel, however, admits that the reading *ilā Qalamūn* is conjectural (*K. al-Fihrist*, p. 193) and says that all the codices read *abī Qalamūn* (without the diacritical point

under the *b*). His emendation to *ilā Qalamūn* is therefore open to question, especially as his further "identification" of *Qalamūn* is very doubtful. In point of fact, there can be little doubt that *abī Qalamūn* is correct: the title would then read "The Book of Abū Qalamūn." Now Abū Qalamūn is the Arabic nickname of the *jasper*, as is shown by Mas'ūdī's description (II, 437) of the "Chatons nommés *baqalamūn*, qui offrent à l'œil des nuances chatoyantes et variées entre le rouge, le vert, le jaune, &c. . . . Le chatolement résulte de l'éclat et de la limpidité de la pierre, et aussi de l'angle sous lequel l'œil la considère." (Barbier de Meynard's translation, Paris, 1863.) See also Ibn al-Baitār, Sontheimer's translation, II, 603 (Stuttgart, 1840): "In the East the jasper (*lit.* the Ethiopian *Yāqūt*) is called *Abū Qalamūn*."

(34) The *Fihrist* mentions a *Kitāb al-BDWH*, which Berthelot misreads as *K. al-Baduh*, leaving the meaning of *Baduh* undecided. The true reading is that given above, viz., *BDWH*. This combination of letters, representing the numerical value 2468, is commonly employed as a kind of talisman to facilitate birth, or as a love-charm, &c. See de Sacy, *Chrest. arabe*, III, 365 (Paris, 1826).

(35) *K. al-Mujarradit*. (*Fihrist*.) "The Book of Extracts." This is no doubt the *Liber Denudatorum* quoted in the *De aluminibus et salibus* ascribed to Rhazes (Al-Rāzī). *Jarrada* may mean "to strip naked" as well as "to make extracts from a book." See Dozy, *op. cit.*, *sub voce*. The *Lib. Denudatorum* is mentioned by Borellius, *op. cit.*, p. 103.

(36) *K. al-Tasrif*. "The Book of Mutation." This seems to be the *Liber Mutatorium* quoted in the *De aluminibus et salibus*. See No. 35, *supra*. Berthelot (*op. cit.*, iii, 34) translates the title "*Livre de la Désinence*," but queries his own translation.

(37) *K. al-Thalathīna Kalima*. "The Book of Thirty Words." A Latin MS., entitled *Liber de XXX Verbis*, anonymous, follows the *Liber de Septuaginta* ascribed to "Geber" in the British Museum MS. *Arundel* 164.

(38) *K. Khamsata 'Ashara*. "The Book of Fifteen." A Latin MS. entitled *Liber XV*, ascribed to "Geber," is in the library of Trinity College, Cambridge (No. 1363, ff. 137 v.-140 v. *sacc.* xv).

(39) *K. Musahhikāt Suqrāt*. "The Book of Emendations of Socrates." (*Cf.* No. 28.) May this be the same as *Ad laudem Socratis dixit Geber?* *Bodl. Ashmole* 1416, f. 148.

(40) *K. al-Sab'īna*. "The Book of Seventy." This has already been fully discussed by Berthelot (*op. cit.*, i, chap. ix, p. 320, and *Archéologie*, Paris, 1906). Other Latin MSS. of the work (*Liber LXX*) occur in the British Museum, *Add.* 10764, fol. 126 v.-127 v., 128-138 v., and *Arundel* 164.

(41) *K. Sharh al-Majista*. "The Book of Comment on the *Almagest*." [*Fihrist*.] A "Comment on Ptolemy," ascribed to Geber, was translated by Gerard of Cremona. MSS. are in Corpus Christi College, Oxford (No. 233, ff. 32-67), the Bodleian (*Ashmole*, 357, ff. 97-178 v.), and Cambridge University Library (Mm. II, 18, ff. 2-49 and I. I, 13, ff. 58 v.-60).

(42) *K. al-Wasiyya*. "The Book of the Testament." British Museum, *Add.* 7722, No. 3. MSS. of the Latin work *Geberi Testamentum* are in Trinity College, Cambridge (925, and 1380 ff. 134-140); the work was also printed many times, e.g., Manget's *Bibl. Chem. Curiosa*, I, 562. I have not compared the Arabic with the Latin, so I cannot say whether the two are identical or not.

(43) *K. al-Mulāgham*. "The Book of Amalgams." Mentioned in the *Rutbatu'l-Hakīm*.

(44) *K. al-Khūlis*. "The Book of Sincerity."

(45) *K. al-Jam'*. "The Book of Collection."

These two (xii and xiii) are mentioned by Jābir in his *Book of Properties* (A. 21,

*supra*). Hoefler (*Hist. de la Chimie*, I, 74), suggested that the *K. al-Khālīs* might be the original of the *Summa perfectionis*, but gave no reason. Steinschneider (*op. cit.*, p. 21) errs in saying that Hoefler quotes Bibl. Nat. *Arabe* 1083 (ancien fonds) as containing a MS. of the *K. al-Khālīs*; the No. 1083 is given by Hoefler as a reference to Hājī Khalifa (ed. Fluegel, 1835-1858). So far as I am aware, no MS. of the *K. al-Khālīs* is extant.

(C) BOOKS NOT MENTIONED IN THE FIHRIST BUT (1) EXTANT, OR  
(2) KNOWN BY TITLE ONLY.

1.—*Extant, complete or fragmentary.*

(46) *Sundūq al-Hikma*. "The Casket of Wisdom." MS. in Royal Library at Cairo.

(47) *Kitāb Ikhrāj mā fi'l-Qauwat ilā al-Fi'l*. "The Book of Extraction from Potentiality to Actuality." MS. Royal Library, Cairo.

(48) *K. al-Hudūd*. "The Book of Definitions." MS. Royal Library, Cairo.

(49) *Kashf ul-Asrār wa Hatku'l-Astūr*. "The Unveiling of Secrets and the Rending of Veils." Brit. Mus. Addl. 7722 No. 4. MS. Royal Library, Cairo. Published with a rendering into English by R. Steele, London, 1892 (Luzac & Co.).

(50) *Risāla (fi'l Kimiā')*. "Letter (on Chemistry)." MS. Royal Library, Cairo.

(51) *K. fi 'Ilm al-San'atīl-Ilāhiya wa'l-Hikmatīl-Falsafiya*. "The Book of Knowledge of the Divine Art and Philosophical Wisdom." MS. Royal Library, Cairo.

(52) *Khawāss al-Iksīr al-Dhahab*. "The Properties of the Elixir of Gold." Paris, Bibl. Nat. *Arabe*, 2625, No. 6. Translated into English by E. J. Holmyard, *Science Progress*, 1922, p. 258, where it is incorrectly stated to be from the *Kitāb al-Khawāss*, No. 21 (*q.v.*).

(53) *Kitāb al-Muqābila wa'l-Mumāthila*. "The Book of Comparisons and Similitudes." Berlin, 4177.

(54) *K. al-Rahma*. "The Book of Mercy." Printed by Berthelot (*op. cit.*, iii) from the Leyden MS. *Arabe* 440. This is really a work of Abu 'Abdullah Muhammad ibn Yahyā containing many quotations from Jābir; the author names himself twice in the course of the book.

(55) *K. al-Rahma al-Saghīr*. "The Little Book of Mercy." Printed by Berthelot (*loc. cit.*) from the Bibl. Nat. MS. 2605. Also lithog., India, 1891.

(56) *K. al-Tajmī'*. "The Book of Concentration." Printed by Berthelot (*loc. cit.*) from the Leyden MS. *Arabe* 440.

(57) *K. al-Tajrīd*. "The Book of Abstraction." Lithog. India, 1891. Jābir says that he wrote this after the "112 Books," and that it forms one of the books of the series on the "Balance."

(58) *K. al-Sahl*. "The Book of Ease." Brit. Mus. Addl. 7722, No. 1.

(59) *K. al-Sūfi*. "The Book of Purity." Brit. Mus. Addl. 7722, No. 2.

(60) *K. al-Ihrāq*. "The Book of Combustion." Quoted by Al-Jildakī in vol. i of his *Nihāyat al-Talab*.

(61) *K. al-Taklīs*. "The Book of Calcination." Quoted by Al-Jildakī (*op. cit.*, vol. ii).

(62) *K. al-Abdāl*. "The Book of Exchanges." Quoted by Al-Jildakī (*op. cit.*, vol. ii).

(63) *K. Zuhr al-Riyāḍ*. "The Book of the Flower of the Garden." Quoted by Al-Jildakī (*op. cit.*, vol. ii).



(64) *K. al-Uṣūl*. "The Book of Roots [= fundamental principles]." Brit. Mus. Addl. 23418, No. 13. Latin translation (?) mentioned by Borellius, op. cit., p. 102. (Liber Radicum.)

(65) *K. Muḥaj al-Nufūs*. "The Book of the Essences of Spirits." Quoted by Al-Jildakī (op. cit., vol. ii).

(66) *K. sharḥ K. al-Rahma*. "The Book of Comment upon the Book of Mercy." Quoted by Al-Jildakī (op. cit., vol. i).

(67) *K. al-Afwā*. "The Book of Pardon." Quoted by Al-Tughra'ī (Brit. Mus. Or. 8229).

(68) *K. al-Rāḥa*. "The Book of Repose," otherwise known as *K. al-Da'āwa*, "The Book of Chains." Quoted by Al-Tughra'ī, loc. cit.

(69) *K. al-Sirr al-Maktūm*. "The Book of the Hidden Secret." Quoted by Al-Tughra'ī, loc. cit.

(70) *K. al-'Awālim*. "The Book of Worlds." Quoted by Al-Tughra'ī, loc. cit. See also *K. al-Mawāzīn*, ed. Berthelot, op. cit., iii, where a work of a similar title is mentioned. There is a MS. of this in Bibl. Nat. *Arabe* 2606.

(71) *K. al-Dhahab*. "The Book of Gold." Bibl. Nat. *Arabe* 2606.

(72) *K. al-Fuḍḍa*. "The Book of Silver." *Ibid*.

(73) *K. al-Nuḥās*. "The Book of Copper." *Ibid*.

(74) *K. al-Hadīd*. "The Book of Iron." *Ibid*.

(75) *K. al-Uṣrub*. "The Book of Lead." *Ibid*.

(76) *K. al-Qaṭī*. "The Book of Tin." *Ibid*.

(77) *K. al-Khārsīnī*. "The Book of Tutenag." *Ibid*. Khārsīnī or Khār of China, often called *Katesim* in Latin translations from the Arabic, is probably an alloy containing zinc, copper and iron. Later on, according to Dozy (op. cit.) the name was applied to zinc itself.

(78) *K. al-Ijāz*. "The Book of Abbreviation." *Ibid*.

(79) *K. al-Hurūf*. "The Book of Letters." *Ibid*.

(80) *K. al-Kabīr*. "The Great Book." *Ibid*.

(81) *K. Nār al-Hajar*. "The Book of Stone." *Ibid*. Also printed by Berthelot (op. cit., iii) from the Leyden MS. *Arabe* 440.

## 2.—Titles only known.

(82) *K. al-Arba'*. "The Book of Four."

(83) *K. al-Taṣṭīd*. "The Book of Sublimation."

(84) *K. al-Ṭyān*. "The Book of Clays."

(85) *K. al-Tanqīyya*. "The Book of Cleansing."

These four books are mentioned, without quotation, by Al-Jildakī in the *Nihāyat al-Talab*.

(86) *K. al-Tanzīl*. The Book of Reduction *per descensum*. Mentioned by Jābir in his "Book of Properties" (No. 21), *q.v.*, and given also in the Leyden codex of the Fihrist.

(87) *K. al-Muntaḥā*. "The Book of the Conclusion." Mentioned by Jābir, loc. cit.

(88) *K. al-Khawāṣṣ al-Khamsīna*. "The Book of the Fifty Properties." Mentioned by Jābir in his *K. al-Zaibāq al-Gharbī* (No. 10).

(89) *K. al-Sumūm*. "The Book of Poisons." Mentioned by Jābir in his *K. al-Mawāzīn* (No. 30), ed. Berthelot, op. cit., iii.

(90) *K. al-Adilla*. "The Book of Indications." Mentioned by Jābir, loc. cit.

(91) *K. Sifat al-Kawn*. "The Book of the Nature of Being." Mentioned by Jābir in the *K. al-Rahmat al-Saghīr*, ed. Berthelot, op. cit., iii.

(92) *K. Tadbīr al-Hukamā' al-Qudamā'*. "The Book of the Operation of the Ancient Sages." Mentioned by Jābir, loc. cit.

N.B.—In concluding this section it ought to be said that the translation of the titles of Arabic alchemical works without previous knowledge of the text is bound to be uncertain.

### (3) AN ESTIMATE OF THE EXTENT OF JĀBIR'S CHEMICAL KNOWLEDGE AND OF HIS CONTRIBUTIONS TO CHEMISTRY.

To appreciate properly the value of Jābir's accomplishments it is necessary to have a general idea of the intellectual atmosphere of Islām in the second century of the Hijra, i.e., the eighth century A.D. In 749 A.D. the 'Umayyad dynasty was overthrown and was succeeded by that of the 'Abbasids. This change ushered in a period of intense literary activity, and, although some translations of Greek scientific and philosophical works had been made previously, it was in the last half of the century that the Muslims first became thoroughly intimate with Hellenic thought. The influence thus exercised soon began to bear fruit, learning was encouraged by the Caliphs, academies and observatories were founded, scholars welcomed, and thousands of books obtained from Alexandria, Byzantium and other seats of ancient culture.

Muslims of a scientific habit of mind turned naturally to philosophy, medicine and alchemy. The first two of these subjects presented no great difficulty, and, moreover, teachers were numerous. Chemistry, however, was in different case. It came to Islām via Alexandria, clothed in mysticism and infected with charlatanry and magic. It was a study despised and often considered to be unlawful; those who interested themselves in it were frequently driven to justify themselves by asserting that the prophets and even 'Alī ibn Abī Ṭālib had practised the art. No idea of chemistry as an independent and reasonable science seems to have penetrated to the intellectual aristocracy of Islām up to the middle of the eighth century.

Into this unfavourable environment Jābir was born. We may well sympathize with him when we consider the superhuman efforts any man of his time would have had to make to clear away the rank growth which surrounded chemistry and to establish the subject impregnably as a science of equal nobility with those of philosophy, mathematics and medicine. Yet this is the task which Jābir undertook and in which he achieved no small measure of success. The cold, impartial outlook which characterizes the man of science of to-day we should not expect to find in him—that is the heritage of centuries of patient progress. Neither should we expect to find in Jābir that calculating materialism and "suspense of judgment" which form so striking a feature of the present scientific age. A man must be judged by the intellectual background of his own time, and if we admit this criterion we shall come to place Jābir on a level with Boyle, Priestley and Lavoisier, as one of the pre-eminent figures in the history of chemistry.

A study of his works shows us that, while primarily a chemist, he was accomplished also in many other directions. Thus he wrote books on medicine; he wrote a commentary on Euclid and on the *Almagest*; he knew some of the writings or views of Plato, Socrates, Aristotle, Pythagoras and Democritus (which

he may have read in the original, as he appears to have known Greek); he wrote a treatise on Mirrors, another on Logic, another on Poetry; he interested himself in the newly-developed system of Sūfī-ism, and he studied the mystical ideas of Apollonius of Tyana. He was thus a widely-read scholar and not a petty mystagogue or charlatan; we can indeed be certain that the Barmekides—a very level-headed family—would otherwise not long have tolerated him.

Fortunately for chemistry, this man of genius and unbounded energy was drawn towards natural science, and, encouraged by the Imām Ja'far al-Sādiq, turned his attention to the study of the composition of substances obtained from minerals, plants and animals. His writings prove that this study meant to him not merely the reading of books but the close investigation of Nature and a stern discipline in the laboratory. It has to be said that Berthelot, having made up his mind—on what seem to be insufficient grounds—that the Latin Geber is not to be identified with the Arab Jābir ibn Ḥayyān, appears deliberately to underrate the latter; he certainly gives an entirely false idea of Jābir's scientific ability. A study of Jābir's works has led me to form a very high estimate of their author's mental calibre, and, whether he be the Latin Geber or no, he certainly had as fine an intelligence and as wide an experience. Some of the material upon which this judgment is based is here adduced.

#### (1) *Nature and Scope of Chemistry.*

Jābir defines chemistry<sup>1</sup> as "a branch of natural science, for it investigates mines and the manner in which, by the action of fire, metals are produced in them, since men desire by artificial means to imitate Nature. Everyone, indeed, who knows anything of [natural] philosophy realizes that the Art [of chemistry] imitates Nature and models itself on her. Now how can a man imitate a thing of which he knows nothing? As for those for whom this book is written, they hold steadfastly to the science therein and know its high place in philosophy."

He who studies chemistry must know that there are ten conditions to be observed for its successful practice. These are (1) the operator should know the reason for performing each operation; (2) the instructions must be properly understood, since every art has its own technical language; (3) the impossible and profitless should not be attempted; (4) time and reason must be carefully chosen [astrological influence]; (5) it is best for the laboratory to be in a secluded space; (6) the chemist must have trusty friends; (7) he must also have leisure to conduct his experiments, (8) and patience and reticence, (9) and perseverance; (10) he must not be deceived by appearances into bringing his operations to too hasty a conclusion.<sup>2</sup>

Although the main problem which chemistry set itself to solve in those days was the transmutation of the metals, Jābir did not allow this to become an obsession with him, and in numerous passages he describes the application of chemical knowledge to what we may term technical processes. The bulk of his writings certainly deal with theories of metallic constitution and are often couched in language which is difficult to understand: he was not completely successful in wresting himself clear from the trammels of mysticism. In spite of this, however, it is abundantly evident to anyone who reads his books that he made valiant efforts to attain to a reasonable scientific method. Where he failed was in trying to arrive at a comprehensive system—like most early scientists he explained too much. He had leanings also to the "number-

<sup>1</sup> *Book of Knowledge of the Divine Art and Philosophical Wisdom, vide supra, No. 51.*

<sup>2</sup> *Op. cit.*

mysticism" of Pythagoras, and his childish delight over magic squares and similar amusements shows that his mathematical ability could not have been very great.

### (2) *Chemical Theories.*

In chemical theory Jābir shows a remarkable advance upon the Aristotelianism and pseudo-Aristotelianism which preceded him. He seems to have accepted in a general way the Aristotelian conceptions of the *prima materia*, the four "elements" and the "four qualities," but he developed more specialized theories of the constitution of metals and in doing so laid the foundations of the phlogiston theory, which, intellectually, is separated by only a short distance from Jābir's own ideas: in time, of course, there is an interval of some eight hundred years between them.

In the *Kitāb al-Īqāh* (*supra*, No. 27, p. 50), he says:—

"The metals are all, in essence, composed of mercury combined and coagulated with sulphur; . . . they differ from one another only because of the difference of their accidental qualities, and this difference is due to the difference of their varieties of sulphur, which again is caused by a variation in the soils and in their positions with respect to the heat of the sun."

The most subtle sulphur is the golden, which, combined with mercury, forms a perfect compound—gold—distinguished from the other metals by the fact that it cannot be burnt but is stable in the fire. This passage is sufficient to show that Berthelot was wrong when he said that the "*œuvres arabes de Djābir n'offrent aucune trace*" of the sulphur-mercury theory of metals, "*théorie que l'on attribue en général à Geber.*"<sup>1</sup> The sulphur-mercury theory, however, appears to have been understood by Jābir not in the literal sense but as an approximation; he knew quite well that ordinary sulphur and mercury when combined gave rise not to a metal but to cinnabar, and therefore the "sulphur" and "mercury" of which metals are composed are not the "sulphur and mercury" of the vulgar, but rather hypothetical substances to which ordinary sulphur and mercury form the closest approximations. The combination of sulphur and mercury gives occasion to some very penetrating remarks by Jābir—remarks which show that he had some idea of the ancient atomic theories, and which, as an expression of his views on the nature of chemical combination, are not merely noteworthy, but astonishing in their clarity and perspicuity:—

"When mercury and sulphur combine to form one single substance, it has been thought that they have essentially changed and that an entirely new substance is formed. The fact is otherwise, however. Both the mercury and the sulphur retain their own natures—all that has happened is that their parts have become attenuated and in close approximation to one another, so that to the eye the product appears uniform. But if one could find an apparatus to separate the parts of one sort from those of the other, it would be apparent that each of them has remained in its own permanent natural form and has not been transmuted or changed. We say, indeed, that such transmutation is not possible for natural philosophers."<sup>2</sup>

### (3) *Chemical Operations.*

Jābir was acquainted with the usual chemical operations such as solution, crystallization, calcination, reduction, &c., and often describes them. Of more interest, however, is the fact that he attempts to understand the changes that go on in these processes and frequently gives his opinion as to their aims. His method of reducing calces is illustrated by the following quotation:—

<sup>1</sup> *Op. cit.*, i, 341.

<sup>2</sup> "Book of Knowledge of the Divine Art and Philosophical Wisdom, *vide supra*, No. 51.

<sup>3</sup> *Kitāb al-Khawāss al-Kabir* (No. 21, *supra*), *maqāla* 38.

"Take a pound of litharge and a quarter of a pound of soda (*qalī*) and powder each well. Then mix them together and make them up into a paste with oil and heat in a descensory. [The metal] will descend pure and white."

On calcination he wrote a book, the *Kitāb al-Taklīs* (No. 61, *supra*, p. 52), from which the passage below is quoted:—

"Souls and spirits [i.e., volatile substances like sulphur and sal-ammoniac] will not sustain calcination, since the latter can be effected only with a very hot fire; now spirits will not sustain a very hot fire as they are volatile and fly away from it. Moreover, the aim of calcination is nothing more than the removal of impurities from bodies and their complete combustion so that the bodies may be purified and remain unadulterated and unsullied; in a spirit, however, there is no necessity for the same treatment as a metallic body, and all that is needed is the first process in calcination [i.e., gentle heating], when the same effect is produced on the spirit as [complete] calcination effects on the metals, namely, full purification. Understand that clearly, therefore. As for the process which is to spirits what calcination is to metals, I swear by my Master that thou wilt find it to be sublimation, and on account of that we have devoted a book to sublimation, following the present book.

"As I have now made clear the aim of calcination I will next speak of its various forms, for each metal is calcined in a different way from the others. This is because among the metals are found some which are already pure, such as gold; in this case the object of calcination is to convert the metal into a fine powder so that it may be enabled to combine and enter into union with the sublimed spirits, and also to dissolve. The same applies to silver, but silver is slightly impure, so that along with the necessity for converting it into a fine powder is also that of purification.

"As for the rest of the metals, that is excluding the two above-mentioned, they indeed all require calcination both for purification and for converting them into powder; and the same is true for those minerals which are infusible, according to their degree of purity."

Two specimens of Jābir's instructions for preparing chemical compounds are appended. They are taken from the *K. al-Khwāṣṣ*, section (*maqāla*) 36.

(a) "Take a pound of litharge, powder it well and heat it gently with four pounds of wine vinegar until the latter is reduced to half its original volume. Then take a pound of soda and heat it with four pounds of fresh water until the volume of the latter is halved. Filter the two solutions until they are quite clear and then gradually add the solution of soda to that of the litharge. A white substance is formed which settles to the bottom. Pour off the supernatant water and leave the residue to dry. It will become a salt as white as snow."

(b) "To convert mercury into a red solid. Take a round glass vessel and pour a convenient quantity of mercury into it. Then take a Syrian earthenware vessel and in it put a little powdered yellow sulphur. Place the glass vessel on the sulphur and pack it round with more sulphur up to the brim. Place the apparatus in the furnace for a night, over a gentle fire . . . after having closed the mouth of the earthenware pot. Now take it out and you will find that the mercury has been converted into a hard red stone of the colour of blood. . . . It is the substance which men of science call cinnabar."

It is fitting to conclude this brief account of Jābir's life and works with his characteristic remarks on experiment:—

"The first essential in chemistry is that thou shouldst perform practical work and conduct experiments. For he who performs not practical work nor makes experiments will never attain to the least degree of mastery. But thou, O my son, do thou experiment so that thou mayst acquire knowledge."<sup>1</sup> "Scientists delight not in abundance of material; they rejoice only in the excellence of their experimental methods."<sup>2</sup>

<sup>1</sup> *Kitāb al-Tajrīd* (No. 57, *supra*, p. 52).

<sup>2</sup> *Kitāb al-Rahmat al-Saghīr* (No. 55, *supra*).



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THE EDITORIAL COMMITTEE

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**VOLUME THE SIXTEENTH**

SESSION 1922-23

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SECTION OF LARYNGOLOGY



LONDON  
LONGMANS, GREEN & CO., PATERNOSTER ROW  
1923



## Section of Laryngology.

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## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### PRESIDENT'S ADDRESS.

By CHARLES A. PARKER, F.R.C.S.Ed.

THE honour you have conferred upon me brings to my recollection the time when the old Laryngological Society was founded, especially as it will complete its third decade during my year of office, for it was founded in 1893. It was fathered particularly by Semon, Butlin, Bowlby, Macdonald, and others. Its first president was Sir George Johnson, who was followed by Sir Felix Semon. I was an original member and attended the first meeting of the Society, and thus I have been able to see how the character of the cases met with and the manner of treating them has gradually changed. Thirty years ago, laryngology was still chiefly the physician's province, reliance being placed on internal medication, and the local application of sprays, paints and powders; but since then the tendency has been for the methods of treatment to become more and more surgical with an ever increasing momentum. When I was resident at the Throat Hospital, Golden Square, in 1889, the beds were chiefly filled with cases of chronic laryngitis, and perhaps some cases of tuberculosis of the larynx; and my duty was to proceed from bed to bed and make local applications to the affected part. The work then was chiefly medical, whereas now practically all the beds in that institution are surgical beds, and surgery predominates. During my residency I never saw a larynx opened, nor a sinus treated otherwise than by puncture; nor did I see a case of acute mastoid treated otherwise than by a Wilde's incision. Yet the staff were not behind the times: Sir Morell Mackenzie, the greatest of pioneers in laryngology, had just retired, and had left his colleagues imbued with the spirit of progress; they were quite abreast of the times. Indeed, they excelled most surgeons of to-day in their intra-laryngeal manipulations by the indirect method, especially in the removal of innocent laryngeal growths. It would be a pity if this piece of craftsmanship were entirely superseded by the direct method in adults as well as children, which is the tendency at the present time.

Thirty years ago laryngology was scarcely regarded as a justified specialty. Now, not only is that changed, but there is even a hope that the Colleges of Physicians and Surgeons may grant a special diploma in oto-rhino-laryngology. And in the time of which I am speaking, it was only London and the larger provincial cities which had laryngologists, whereas to-day it is a specialty well to the fore even in the smaller towns and many country districts. In

those early days, even the general hospitals looked upon laryngology as of quite secondary importance, and if they had a special department at all, it was staffed by a general physician or an assistant-surgeon. Now every important hospital has a good special department staffed by laryngologists in every way on an equality with the rest of the hospital work.

I think that this Section and the old Laryngological Society have played an important part in bringing about these changes. By their efforts and by their printed *Proceedings* it has been proved to the world that laryngology is a science requiring such a wide field of special knowledge, and such a special technique, that no one now questions that it has justified itself as a specialty. I agree with what Sir William Milligan said as to the Section being an educative body. Younger men, being perhaps for the first time in charge of a clinic, learn by bringing cases and hearing them and others discussed, and perhaps specially do they learn when views on their cases contrary to their own are expressed. It is easy for all, including the senior men, to fall into a rut, and the chief safeguard against that is to meet and rub shoulders with colleagues and to learn what progressive ideas are germinating in the minds of others. But for this, the practice at special hospitals and departments is liable to become stereotyped and lack the spirit of progress. Thus, both seniors and juniors must benefit from having a common meeting-ground. It has been said that each generation starts where its predecessor leaves off, but in this specialty, each man starts where his colleague leaves off, and it is at meetings of this Section that we learn the latest which has been thought and done, and we receive the stimulus to new ideas which in their turn carry us still further onwards. It is by meeting together and learning from each other in this way that the great progress of the last thirty years has come about. Little by little we have led each other on from one small advance to another, and the sum total of these small advances has meant a revolution in the science and art of laryngology to the great benefit of suffering humanity.

### Suppurating Dental Cyst. Drained: Subsequently obliterated by the Blood-clot Method.

By DAN MCKENZIE, M.D.

PATIENT, a male, aged 45, in March, 1922, with a history of pain in the right cheek for a week and terminating in a discharge of pus and blood from the nose. At a later date during a second attack of pain and swelling, I was able to confirm his observation that pressure on the swollen cheek made pus emerge from the nose, which was found to proceed from an ill-defined swelling, involving bone, in the floor of the right vestibule. The right antrum was dull on transillumination, but as the right half of the vault of the hard palate was flattened, a diagnosis of suppurating dental cyst was made.

A few days later an incision made for drainage in the gingivo-labial recess of the upper lip confirmed the diagnosis. In May, the suppuration having subsided, the cyst was freely opened and the lining membrane thoroughly removed. But no attempt was made to remove the delicate bony wall surrounding the cyst which occupied most of the right antrum and extended in the hard palate across the middle line. On the other hand, the cavity was preserved and allowed to fill up with blood and blood clot and the buccal wound was entirely sutured up. The result has been satisfactory.

## DISCUSSION.

Mr. W. STUART-LOW said he agreed with the treatment in this case. He had operated on a number of such cases—one quite recently—by excising the cyst and suturing up. In the latter case he thoroughly curetted it out. He disapproved of prolonged packing as likely to cause a persistent sinus.

Mr. D. L. SEWELL remarked that he had had three cases in which he experienced great difficulty in closing the opening into the buccal cavity, following the usual opening through the cyst wall into the antrum, and from the antrum through the meatus into the nose. In one case a plastic operation was necessary to close the opening.

Mr. LAWSON WHALE asked what Dr. McKenzie and Mr. Stuart-Low would have done in a case in which the course advocated was impossible, because the cyst had passed through the socket of the canine into the nasal fossa. When he had opened the cyst from below, a probe passed into the nose. He had been unable to do anything but pack; fortunately the cavity had healed.

Mr. E. D. D. DAVIS said he had treated thirty-one dental cysts. Eight had been incised, curetted and packed before he had seen the cases, and as soon as the opening had closed, the cyst had re-formed. Complete excision from the alveolus had been carried out with success. As all the large dental cysts which he had seen had been suppurating and of long duration, he had not adopted Dr. McKenzie's method, though he intended to do so in future. It was very difficult to obliterate the cavity of a large dental cyst which bulged into the nose and antrum, especially when it extended into the hard palate. Dental cysts, particularly the palatal type, were situated well below the floor of the nose, and drainage into the nose was in most cases unsatisfactory; a persistent fistula sometimes occurred between the mouth and nose when such drainage was established, though careful suturing of the mouth wound might prevent the formation of a fistula. For the reasons above stated he preferred to enucleate the cyst from the mouth and carefully avoid opening into the nose.

Mr. E. M. WOODMAN asked what happened when the permanent teeth were implicated in the cyst. He had had a case of a large cyst which was full of thick pus; the roots of the teeth were projecting into the cyst, and were suppurating. But for those teeth he would have employed the blood-clot method. Two definite fistulae in the mouth were left. He drained the cyst, but spontaneous closure did not follow, and therefore he had to pack the cavity.

Dr. MCKENZIE (in reply) said that an interval of two weeks had elapsed between the first and second operation. It was the second case he had treated by this method. The first was not such a large cyst, but it healed up satisfactorily. With regard to openings which became permanent, a method had been described in a French paper of cutting a flap from the inner surface of the cheek, and closing the opening with it. He did not know whether the formation of fistulae would be so likely if all cases were treated by the blood-clot method. The origin of these cysts was unknown. Some had no obvious connexion with the teeth; in some an unerupted tooth was embedded in the cyst wall, whilst in others the roots of permanent teeth were laid bare, doubtless by expansion of the cyst wall. He did not know whether the blood-clot method would answer with very large cysts; at all events he considered it worthy of an extended trial.

### The Timeous Treatment of the "Broken Nose."

By DAN MCKENZIE, M.D.

PATIENT, a male, aged 21, is shown to illustrate how the deformity of a broken nose can be easily and permanently rectified if treated by suitable manipulation before the fractures have had time to unite. He was first seen in July of this year, following the receipt of a blow on the bridge of the nose

five days previously. The bridge was swollen, but in spite of the swelling it was seen to be displaced towards the left, while palpation revealed that the right nasal bone was tilted so that its lower edge formed a prominence under the skin. Internally the cartilaginous septum presented a hard vertical bulge: this showed that it also had been fractured or buckled.

Under chloroform, by means of external manipulation aided by a flat padded elevator inside the nose, the deformities were easily reduced. With the exception of a padding of gauze inserted for twenty-four hours in the atrium of the nose under the bony bridge no apparatus was applied. The result is a straight nose. There is, however, still a depression on the nasal bridge just below the ends of the nasal bones. This is due to the septal deformity which still exists to some extent.

### **Injury to the Nose from a Lift Accident.**

By W. M. MOLLISON, M.Ch.

PATIENT, aged 21, fractured both superior maxillæ and mandibles about a year ago. In January, 1922, he was suffering from nasal obstruction and there was a sinus at the root of the nose. Many crusts were present in the nose and naso-pharynx and bare bone was felt through the sinus.

Six months ago removal of several bony sequestra through the nose and the sinus led to much improvement.

Three weeks ago the operation of inserting a cartilage inlay to build up the nose was undertaken. On raising the soft tissues from the very depressed scar of the old sinus an opening was made into the nose: and suppuration followed. In spite of the suppuration a piece of cartilage was inserted to close the hole, and it is interesting to find that the cartilage has taken.

### **Case of Depressed Bony Bridge of Nose.**

By H. D. GILLIES, C.B.E., F.R.C.S.

THE patient shown, Miss R., presents an example of depressed bony bridge of nose following a blow, and probably septal abscess. Operation: Reconstruction by cartilage implantation (*see* photographs, p. 5).

### **Depressed Fracture of Nasal and Associated Bones.**

By H. D. GILLIES, C.B.E., F.R.C.S.

PATIENT, a female, Miss G. Photograph presents an example of depressed fracture caused by motor accident, in which not only the nasal bones but the associated bones which form the base of the nasal arch have been driven backwards. Attempt at replacement by elevation of bones and support by (Carter) bridge, a failure. Later, correction of deformity by cartilage graft.

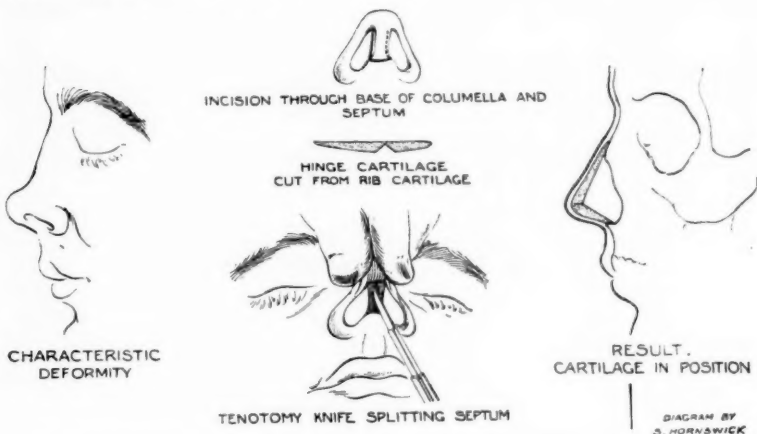


Case of depressed bony bridge of nose.

**Case of Depressed Fracture of Nasal Arch.**

By H. D. GILLIES, C.B.E., F.R.C.S.

PATIENT, a male. Photograph shows an example of old depressed fracture of the nasal arch. Replacement of bones is of little value in these cases. Operation: Reconstruction by cartilage graft, which has greatly improved appearance and nasal airway.



The hinged cartilage operation for nasal support. The perichondrium is left continuous across the hinge.

**DISCUSSION.**

Mr. CHARLES A. PARKER (President) said the results in Dr. Dan McKenzie's case were very good, but the interior of the nose needed further attention, as there was much chronic hypertrophy of the turbinates, and the septum was not quite straight. It was often possible to get the nasal bones in good position, and also to straighten the septum, but the difficulty was to prevent the deformity of the latter from recurring; splints and packing seemed to fail. Often, after such injuries, perichondrial thickening occurred, which went on increasing for some time after the accident, and led to nasal obstruction, which eventually necessitated submucous resection.

Mr. H. D. GILLIES said it was necessary to distinguish between pure lateral displacement and displacement with depression. They were easy to manipulate into position if the fracture was the first the patient had had and there was an opportunity of treating it within a fortnight of the accident. After that time he found it necessary to incise at the pyriform opening into the vestibule, and chisel where the bone was fractured, and then reset the bony arch. No splints were necessary. The bone should be mobilized, and the nasal bones raised, and the sides of the nose should be pinched in. If there had previously been deformity of the septum, it would not be cured in that way. If the deformity was due to fracture of the septum, the septum should first be resected, and after that the nose reset. If there had been several fractures to the face, he found that no manipulative work was effectual, and that resort had to be made to the use of a graft.

Mr. W. STUART-LOW recommended the persistent application of cold in these cases, continued for hours after the occurrence of the fracture. This prevented effusion into the nose and tissues, and so avoided subsequent thickening.

Mr. A. J. M. WRIGHT said that in some cases he had been able, by exerting considerable force, to get the bones back into position within three or four weeks of the accident. On three occasions he had used a cartilage graft from the septum where there was not extreme depression, and the result had been quite satisfactory.

Sir JAMES DUNDAS-GRANT said that in cases in which the nose was driven to one side, placing a fold of lint on the other side of the nose and hammering with a mallet was sometimes very effective. In regard to "timeous-ness," he referred to a case in which he had quickly re-set the broken nose of a rider pitched on to his pony's head at polo (he had been rendered unconscious) before consciousness returned.

Dr. MCKENZIE (in reply) agreed that it was important to get these cases before a fortnight had elapsed. If general surgeons and practitioners would only recognize that fact, they would probably send cases to the rhinologist early, when they could be dealt with successfully.

Mr. GILLIES, in reply to Mr. Mollison's query, said that in his war cases cartilage sometimes became infected, but if well drained some of the cartilage might survive. He considered that in the case exhibited by Mr. Mollison, the face should have been brought forward first and the nose re-modelled afterwards.

### **Submaxillary Gland containing a large Salivary Calculus.**

By DAN MCKENZIE, M.D.

THIS submaxillary gland was removed from a patient from whom I had on two previous occasions removed large salivary calculi. The gland was removed at the patient's request and a third calculus, the presence of which was unsuspected, was found in it after removal.

A large salivary calculus removed from the gland was also exhibited.

#### **DISCUSSION.**

Mr. CHARLES A. PARKER (President) exhibited a specimen of a salivary calculus which he had removed from a female patient several years ago.

Mr. A. A. SMALLEY said he had also recently removed a fairly large salivary calculus from a patient who was thought to have malignant disease.

Mr. NORMAN PATTERSON said that in some cases two calculi were present, therefore when one was removed it should be carefully examined as sometimes a smooth facette was discovered which gave the clue to the existence of a second stone.

### **Case of Papillomata of the Trachea.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D., and  
J. J. PERKINS, M.B.

PATIENT, a boy, aged 14: had always been delicate. No chest trouble until two years ago when he caught a cold which was followed by gradually increasing dyspnoea. Admitted two months later to general hospital with loose cough and non-offensive sputum—occasionally streaked with blood. No history of foreign



## 8 Dundas-Grant: *Epithelioma*; Ridout: *Laryngectomy*

body; no tubercle bacilli. Marked inspiratory dyspnoea. Retraction of sternum and ribs along line of Harrison's sulcus; dullness right base; diminished breath sounds; signs, posteriorly, of dilated tubes; expiratory rhonchi over rest of lungs. Seen first by exhibitor on August 3, when a large lobulated fleshy growth was seen, forced up from below the right vocal cord during expiration. A diagnosis of fibroma of the trachea was made. August 5: Attempts at removal by suspension laryngoscopy failed on account of the dyspnoea, so tracheotomy was performed. Through the tracheotomy opening the trachea was seen to be almost completely filled by papillomata, which were removed through a bronchoscopic tube passed through the tracheotomy wound. August 10: A further papilloma was removed in same manner, since it could not be reached by suspension laryngoscopy. August 11: A few papillomatous remains were treated by calcium and magnesium internal medication. October 3: Larynx clear and all symptoms disappeared.

Section of growth showed typical papilloma.

Mr. CHARLES A. PARKER (President) said that this case illustrated the progress which had been made in the direct examination of the respiratory passages. Before the introduction of suspension laryngoscopy, he had attempted removal of a multiple papilloma from the trachea through a tracheotomy wound, and had found how difficult it was to do anything with precision without a view of the growths.

### Case of Epithelioma of the Right Half of the Fauces treated by Diathermy.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

MALE, about 50 years of age, complained of great pain shooting up to the right ear, worse at night, and of increasing difficulty in swallowing. There was a "cauliflower" growth on the anterior pillar of the tonsil, most marked, on the posterior pillar, where the hardness on palpation was very considerable; there was an extensive area of congestion on the right half of the soft palate. The growth was treated by diathermy in July. His symptoms have subsided. In this, as in several other similar cases, there is a history of excessive smoking of strong tobacco, the disease developing on the side of the pharynx opposite to that in which the pipe was held.

Dr. DAN MCKENZIE, commenting on the remark in the notes about excessive smoking, said that he had tested his own mouth during smoking, with a thermometer in the line of the tobacco smoke, but to his surprise the mercury did not rise above 96° F., so that if tobacco smoking was ever the cause of cancer, the exciting agent must be the nicotine and not the heat.

### Case of Laryngectomy following Thyro-fissure.

By C. A. S. RIDOUT, M.S.

PATIENT, a male, aged 51, first seen in February, 1922, complaining of hoarseness and loss of voice for two months.

On examination the upper surface of an ulcerated growth of the left vocal cord was seen, extending to and just involving the left false cord. Move-

ments of both sides of glottis unimpaired. Carcinoma was diagnosed and laryngo-fissure was performed on February 14, 1922, when a much larger mass on the left side, than seemed probable from previous examination, was found penetrating below the glottis. The growth was widely removed by sub-perichondrial resection, and it was believed that a clear margin free from growth was obtained. No cartilage was removed. Patient did well for a time except for a slight stitch suppuration in the wound.

In July, 1922, slight dyspnœa commenced and a warty granulation appeared at the site of the stitch sinus, and ulceration on the interior of the left thyroid ala could be seen with the laryngoscope. Laryngectomy was performed August 29, tracheotomy having been carried out ten days previously. The operation of laryngectomy was rendered very difficult by:—

- (1) Scarring of previous tracheotomy in February.
- (2) The presence of extruding granulations in the site of the old wound.
- (3) The short thick neck of the patient.

It was found impossible to bring the severed end of the trachea up to the skin. The left ala of the thyroid was found much involved in growth which had penetrated it and was already involving muscles on the thyroid surface. A wide clearance of these structures was therefore made. After-progress uneventful.

Pathological report of tumour: Squamous-celled carcinoma.

The specimen is also shown.

#### DISCUSSION.

Dr. P. WATSON-WILLIAMS said anyone who obtained such results as those of Mr. Ridout, was to be congratulated. It was erroneously assumed by some that a man who had undergone laryngectomy and left with no voice was of necessity miserable, but this man, who was really very cheerful, did not bear out such a view. If laryngectomy gave a patient a chance, he should have that chance, and the view that life was not worth living without a voice should be discouraged.

Mr. RIDOUT (in reply) said that he had received several hints from members as to making the man's life more comfortable by enabling him to speak, and he hoped later to try one or more of them.

### **Specimen of Carcinomatous Larynx removed by Laryngectomy.**

By C. A. S. RIDOUT, M.S.

A FEMALE, aged 60, first seen September 9, 1921, with marked dyspnœa. Examination revealed extensive intralaryngeal growth with fixation of all structures on right side of larynx. Patient, a very nervous woman, was advised to undergo early operation, and laryngectomy was performed on September 11, preceded immediately by tracheotomy owing to breathing becoming seriously embarrassed on administration of anæsthetic. Patient's after-progress was most satisfactory and she is alive and well at the present time, one year after operation.

The growth proved, microscopically, to be a squamous-celled carcinoma.

## 10 Ridout: *Parts removed Post Mortem in Tracheal Obstruction*

### Parts removed Post Mortem in a Case of Tracheal Obstruction.<sup>1</sup>

By C. A. S. RIDOUT, M.S.

THIS specimen was obtained at autopsy from a boy, aged 16, who suffered from dyspnoea, and the patient died in May, 1922, from septic pneumonia. The notes of the case were discussed on December 2, 1921.

The specimen shows marked enlargement of both lobes of the thyroid gland, together with enormous enlargement of the thymus, which consists of large discrete lobules clustering around the bronchi. Looked at casually the trachea and bronchi appear very slightly if at all abnormal, but on examination of the cartilaginous rings of the trachea and bronchi it is found that instead of the cartilage forming two-thirds of a circle, in reality it only exists in the anterior one-third; this evidently gave rise to the extreme flattening from side to side found at operation. The condition is not confined to any one portion of the trachea and bronchi but is practically the same throughout. Thyroid, weight 147.5 grm., showed great enlargement in all dimensions. Microscopically, enlargement of vesicles and excess of colloid material. Thymus enlarged in all dimensions, divided into three lobes. Microscopically, showed absence of normal number of Hassall's corpuscles, particularly in the section of the median lobe. Suprarenal: Normal size, no microscopical abnormality.

#### DISCUSSION.

Mr. CHARLES A. PARKER (President) said the specimen showed that the dyspnoea during life was probably due to pressure on a mal-formed trachea, but the question whether the deficiency in the tracheal rings was due to a congenital defect or to an arrest of development after typhoid fever still remains unsettled.

Mr. G. W. DAWSON said he had seen several cases in which a thyroid tumour pressing on the trachea had caused atrophy of the rings of the trachea. On taking a deep inspiration, the trachea was sucked in, so that in most cases it was advisable to employ intratracheal ether.

Sir JAMES DUNDAS-GRANT asked whether, after the removal of the pressure from the trachea in these cases of "scabbard" trachea, the trachea ever recovered a circular form? He thought not.

Dr. IRWIN MOORE said he had discussed this specimen with Professor Shattock, who had concluded that the want of cartilage was due to atrophy from pressure of the enlarged thyroid and thymus glands. Mr. Ridout had presented the specimen to the Museum of the Royal College of Surgeons, and he (Dr. Irwin Moore) hoped to receive a report later from Professor Shattock for publication in the *Proceedings*.

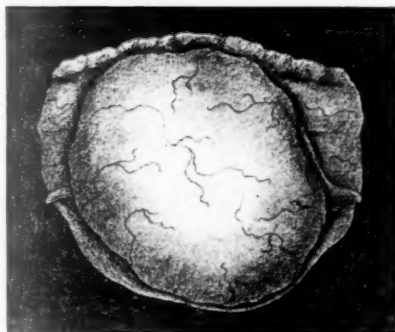
Mr. RIDOUT (in reply) said that the post-mortem appearance was very different from that seen during life when he performed tracheotomy. In the former case the trachea was collapsed, like a piece of flaccid rubber, whereas during life its lumen appeared to be much smaller than its actual size. The thymus gland (as seen in the specimen) was enormously enlarged, and the abnormality of the tracheal rings extended to the bronchi. There were some enlarged bronchial glands below the thymus.

<sup>1</sup> See *Proc. Roy. Soc. Med.*, 1922, xv (Sect. Laryng.), p. 13.

### Lipoma of the Larynx removed by Operation.

By A. J. M. WRIGHT, F.R.C.S.

GROWTH removed from right ary-epiglottic fold of a male, aged 45, under general anæsthetic and suspension laryngoscopy. Symptoms had been present for five years or more, and consisted only of a feeling of dryness and discomfort in the throat and thickness in speech.



Lipoma of the larynx.

### Submucous Lipoma in the Glosso-epiglottic Furrows.<sup>1</sup>

Shown by T. B. LAYTON, D.S.O., M.S.

REMOVED after death from a man, aged 76, who, after entering a restaurant made an incoherent noise and a motion with his hand, which was construed as being a request for water, and then suddenly fell dead.

#### DISCUSSION.

Mr. J. F. O'MALLEY referring to the rarity of lipomata of the larynx, said that when he looked up the literature a few years ago he could find records of only thirteen cases. Various tumours grew at the lower part of the pharynx and base of the tongue which were not entitled to be classed among lipomata of the larynx, as the latter usually grew in the ary-epiglottidean fold. In a case of his own the patient had at times very distressing dyspnoea on undue exertion. The lipoma could be seen dropping down between the vocal cords and acting as a ball-valve. If it had been allowed to remain there much longer the patient might have been suffocated.

Dr. IRWIN MOORE reminded members of a contribution on this subject published in the *Proceedings* of the Section of Pathology, by Professor Shattock,<sup>2</sup> on a large

<sup>1</sup> Specimen No. 1271.1, from the Museum of the Royal College of Surgeons.

<sup>2</sup> "Large Laryngeal Lipoma of the Epiglottis and Base of the Tongue, with a Collection of Examples of Submucous Lipomata of the Intestine and of the Larynx," *Proc. Roy. Soc. Med.*, 1909, ii (Path. Sect.), pp. 285-296.

lipoma of the glottis and base of the tongue. Dr. Irwin Moore also showed a specimen of lipoma of the larynx which was removed by Mr. Hunter Tod.<sup>1</sup>

Mr. E. M. WOODMAN asked for opinions as to the reason why these patients with respiratory obstruction died so quickly. Ten days ago a patient was having four teeth extracted in a Birmingham dental surgery and suddenly died. Strychnine and ether were administered, the abdomen quickly opened, and the heart massaged, without avail. At autopsy a small mop was found tightly wedged in the trachea. If this had been suspected, other steps would have been taken. Was the sudden death due to fright, to shock, or what?

Mr. T. JEFFERSON FAULDER thought that on account of the rarity of lipomata all specimens should be recorded. He had had a case of lipoma of the retropharyngeal space, which was more than 6 in. long, and it had been mistaken, previous to operation, for a retro-pharyngeal abscess. It was removed through the posterior triangle of the neck.

Mr. A. J. M. WRIGHT (in reply) said that notwithstanding the size of the specimen, in his case no dyspnoea was present and the symptoms were trivial. The tumour was very easily removed.

### Papilloma of Septum Nasi.

Shown by H. LAWSON WHALE, F.R.C.S.

REMOVED from the cartilaginous part of the septum of a male, aged 46.

Mr. LAWSON WHALE referred to the rarity of the tumour and compared its size to that of a man's thumb-nail.

### Case of Tuberculosis of the Larynx, with Demonstration of Instrument for Sunlight Treatment.

Shown by Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

MALE, aged 58, was first seen in June, 1920, suffering from hoarseness and cough, and loss of voice of seven weeks' duration. An elongated swelling of the posterior half of the right vocal cord was seen, on which was a shallow ulceration, while the opposite vocal process was in a pachydermic condition. On the first examination no bacilli were found, but a second examination of sputum coughed up after sniffing ol. sinapis, showed bacilli to be present. The galvano-cautery point was into the right vocal cord at the site of the ulcer.

Patient has since been in the south of France and for the last three weeks he has been having sun-treatment by means of a metallic laryngeal mirror held in a frame devised by Dr. Kowler, of Mentone, which the patient brings with him.

When seen on October 17 last, the right cord appeared nearly normal but on subsequent inspection a small slit was detected on the upper surface and a slight degree of pachydermia of the left vocal process. The patient appears to be in perfect health.

<sup>1</sup> "Lipoma of the Larynx," shown at a meeting of the Section of Laryngology, Dec. 7, 1917, see *Proc. Roy. Soc. Med.*, 1918, xi (Sect. Laryng.), p. 67. For drawing see "A Description of the more Interesting Specimens from the Pathological Museum of the First Annual Summer Meeting of the Section of Pathology," 1919, *Proc. Roy. Soc. Med.*, 1920, xiii (Sect. Laryng.), p. 77.

**Case of Ulceration of the Palate and Fauces.**

By W. H. KELSON, M.D., and W. H. THORNHILL, M.D.

PATIENT, a male, aged 64, was first seen a year ago complaining of swelling and congestion of the palate and fauces: the condition remained stationary for some months until June, when ulceration of the soft palate and uvula was observed. The uvula has now disappeared. No membrane was present and there was no pain. Liquids sometimes passed through the nose and occasionally food was regurgitated, but œsophagoscopy revealed nothing abnormal. In appearance the condition resembled epithelioma, but portions removed for examination failed to confirm this (slides exhibited). There is no history of syphilis and Wassermann's reaction is distinctly negative: slight enlargement of the cervical glands can be detected.<sup>1</sup>

Opinions are invited as to diagnosis.

**DISCUSSION.**

Mr. TILLEY had no doubt that this was a malignant condition.

Mr. CHARLES A. PARKER (President) said it would be well to hear the result of further investigation. He thought it was chronic tuberculosis, or lupus.

Dr. W. H. KELSON replied that he did not agree with Mr. Tilley's view; the sections, exhibited in the room, showed no epithelial growth at all. It was very slow and painless, and looked like lupus.

*Report on the Microscopic Section by Professor S. G. Shattock, F.R.S.—*  
"The lesion is a chronic inflammatory one, showing proliferation of connective tissue cells, accompanied by the presence of plasma cells and lymphocytes, without polymorphs. It contains no giant cells."

A section will be stained for tubercle bacilli, and the result will be reported later.

**Myeloid Sarcoma of the Posterior Pillar of the Fauces.**

By NORMAN PATTERSON, F.R.C.S.

PATIENT, a male, aged 50, who four years ago noticed a small swelling on the right side of the neck, movable but not painful. He could manipulate it and cause its disappearance for a few days. Latterly he has noticed that fluid escapes from his nose after drinking if he holds his head forward. He also commenced to spit up a little blood in the morning. For some time he has noticed a discharge coming into the throat in the morning. He has lately been losing weight. When first seen a few weeks ago, a large, soft, freely movable tumour, of a reddish-blue colour, and somewhat irregular surface, was observed growing from the neighbourhood of the right posterior faucial pillar. The teeth were in a very septic state. On October 14 the teeth were extracted, and a portion of the tumour was removed for microscopic examination. Report: A myeloid sarcoma. Section shown.

<sup>1</sup> A von Pirquet test has been carried out and is negative.

Samples of Bismuth and Glycerine Gauze for packing the Nose, particularly after Septum Resection, were exhibited by Sir STCLAIR THOMSON, M.D.

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THE following cases or exhibits have been referred, along with the discussion, for later publication, until further investigations or completed reports have been submitted :—

- (1) H. BELL TAWSE, F.R.C.S. : (a) "Laryngeal Case for Diagnosis ; Swelling of Ventricular Bands." (b) "Ulceration of the Left Tonsil—for Diagnosis."
- (2) T. JEFFERSON FAULDER, F.R.C.S., and F. C. ORMEROD, F.R.C.S. : "Swelling of the Right Cheek—for Diagnosis."
- (3) T. JEFFERSON FAULDER, F.R.C.S. : "Sub-glottic Growth."
- (4) Sir JAMES DUNDAS-GRANT, M.D. : "Case of Ventricular Band Phonation simulating Tuberculous Laryngitis."



## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### Case of Shrapnel-wound of Larynx.

By JAMES ATKINSON, M.B.

PATIENT, a male, wounded 1918, by piece of shrapnel which entered the neck on the left side and passed out on the right; both wounds close to level of upper border of the thyroid cartilage. He suffers from hoarseness varying in degree.

The exhibitor would like to have opinions of Members of the Section as to what is seen in the laryngeal image.

#### DISCUSSION.

Mr. CHARLES A. PARKER (President) said there was some paresis of the inter-arytenoid muscle, and the tips of the vocal processes were very prominent. If there had not been the history of the shrapnel injury he would have regarded the condition as chronic laryngitis, with some paresis of the cords.

Mr. NORMAN PATTERSON said he considered the larynx proper was practically normal, except for some swelling of the anterior portions of the ventricular bands. But there were swellings above the larynx—that on the left side being more marked—and they probably represented the greater cornuæ of the hyoid bone. He thought a skiagram, showing both antero-posterior and lateral views, would be useful in interpreting this most interesting laryngeal picture.

Mr. MARK HOVELL said that the swellings were probably present before the accident.

Dr. JOBSON HORNE remarked that the appearance was unlike anything he had seen, and he did not feel sure that the condition was caused by shrapnel.

Sir STCLAIR THOMSON remarked that if there had not been the history of the injury, he would not have considered there was much the matter with the larynx, except that the patient had ventricular band speech, and the internal tensors of his cords were defective. He advised re-education of the voice. The missile, he thought, had gone through the ventricular bands.

Dr. ATKINSON (in reply) said the larynx showed an ordinary picture of slight chronic laryngitis; but this was merely incidental. The predominating interest in the case was the displacement of the cornuæ of the hyoid bone—seen just above the arytenoids—due to contraction of the thyro-hyoid membrane, or fracture of the greater cornuæ of the hyoid bone, and resulting from the wound. There was no evidence at all of any injury to the ventricular bands.

## An Unusual Tonsillar Appendage and its Relation to Cartilage Formation in the Tonsil: Lantern Demonstration.<sup>1</sup>

By A. LOGAN TURNER, M.D.

(ABSTRACT.)

THE tonsils, in this case, were removed from an adult male by Captain E. D. Dalziel Dickson, R.A.M.C., and sent to the writer for examination. Their histology was studied by Dr. Thomas Sprunt, of the Pathological Department of the Royal Infirmary, Edinburgh. The right tonsil showed no abnormality, but there projected downwards from the left tonsil an appendage measuring 2.5 cm. in length and 0.7 cm. in its greatest transverse diameter. The appendage consisted of a core of fibrous tissue in which were embedded a number of cartilage islets. The preparation supported the view that cartilage in the tonsils was of embryonic origin and did not result from a metaplasia of the connective tissue.

### DISCUSSION.

Dr. WILLIAM HILL said it had been stated that cartilage cells were normal in the fibrous frame-work of the faucial tonsils, especially in the young. Therefore there was no need to invoke any special embryonic departure from the normal to account for the presence of the cartilage itself. Abnormal developments of these cartilaginous areas were rare.

Dr. IRWIN MOORE said that this contribution was of great interest to him, as he was shortly publishing a paper on the subject. He thought the pedunculated cartilaginous tumour now shown was unique. He confirmed the opinion that cartilage cells were always found in the connective tissue structure of the tonsil, and never in the lymphoid tissue, and that these cartilaginous islands were surrounded by perichondrium. Again the cartilage was always of the embryonic type. Orth (Göttingen)<sup>2</sup> first observed cartilage in this situation in 1893, and Walsham<sup>3</sup> and Wingrave<sup>4</sup> in 1898. The latter concluded that it was present in only  $\frac{1}{4}$  to  $1\frac{1}{2}$  per cent. of cases. The recent research work of Mantchik,<sup>5</sup> of Geneva, has shown that cartilage was present in 38 per cent. of the cases he examined. He (Dr. Irwin Moore) exhibited by means of the epidiascope, microscopic sections, recently prepared by Professor S. G. Shattock, showing cartilaginous islands in the capsule of a specimen of lympho-sarcoma of the tonsil removed by Mr. Howarth, also similar islands in the connective tissue capsule surrounding the ivory exostosis recently recorded by Mr. Tilley.

Mr. J. F. O'MALLEY remarked that the process in which Dr. Logan Turner found the cartilage was on the free surface of the tonsil—that which projected into the pharyngeal space. One would often expect cartilaginous inclusions on the capsular aspect, if one reflected upon the development of the tonsil and its relationship with the branchial arches. That part of the second arch designated the epi-hyal was the portion brought into contact with the second cleft, from which the tonsil developed. This case seemed to be one in which cartilage was displaced far beyond the point at which one would expect to find it, and the case was therefore of exceptional interest and rarity.

<sup>1</sup> This contribution will be published in *extenso* in the *Journal of Laryngology and Otology*.

<sup>2</sup> Festschrift zu Virchow's "Fünfzigjähriges Doctorjubiläum," Göttingen, 1893. Ueber Metaplasie, XVI Congress I. M., 4B Section.

<sup>3</sup> *Lancet*, 1898, ii, p. 394.

<sup>4</sup> *Lancet*, 1898, ii, p. 750; also *Proc. Laryngol. Soc.*, 1898, v, p. 34.

<sup>5</sup> *Archiv. Internat. de Laryngol., Otol., Rhinol.*, 1922, n.s. xxviii, p. 257.

Dr. LOGAN TURNER (in reply) said this was the only specimen of the kind he had seen, and apparently no members had previously seen a similar one. The embryonal inclusion theory was the most satisfactory explanation of the occurrence of cartilage in the tonsil.

### Case of Myasthenia Gravis in which Throat Symptoms were an Early Sign.<sup>1</sup>

By C. P. SYMONDS, M.B.

(Introduced by Mr. T. B. LAYTON.)

FEMALE, a school teacher, aged 44. First complained of ptosis, when tired, at the age of 21. In April, 1919, whilst teaching her voice became nasal just as if she "had a cleft palate," and she was unable to clear her throat properly. Subsequently dysarthria, difficulty in chewing and in swallowing. All these symptoms cleared up from time to time. Later she had double vision, and at times lost power in the upper and lower limbs. She has been carrying on her work without intermission from September, 1919, up till September, 1922, though from time to time she has been working under considerable difficulty owing to her symptoms. At present the incapacity for sustained effort is most marked in the upper eyelids.

The symptoms in the early stages were such that the patient was twice referred to a laryngologist for an opinion. The characteristic features of myasthenia gravis in the early stage were present, as there were marked remissions of the symptoms, and these only occurred on fatigue.

From the point of view of the neurologist, it is interesting that this patient's condition should have been repeatedly diagnosed as hysteria. It is time that hysteria ceased to be a dumping-ground for unsolved diagnostic problems, especially since—by a proper mental examination—positive evidence of the disease, when present, can be discovered.

#### DISCUSSION.

Mr. E. D. D. DAVIS said he had seen three cases of the kind, in addition to those shown at the Section. These patients usually came to the laryngologist on account of a defect of speech. His cases were fairly advanced, and the patients had a marked nasal voice typical of a paralysis of the soft palate, with regurgitation of fluids through the nose. On examining the palate, it was seen that the paralysis became more and more marked as the patient became fatigued. The larynx showed a definite adductor chink as seen in functional aphonia and, on inducing fatigue, the cords became immobile and resembled abductor paralysis, also difficulty in swallowing was experienced. The variable paralysis with the expression of the face was characteristic, and if accompanied by ocular paralysis the diagnosis was complete. He asked as to prognosis. Some patients suffered from attacks of dyspnœa. Mr. Somerville Hastings had shown such a case in which death occurred suddenly from dyspnœa. One of his own cases remained in much the same condition for two years and then was lost sight of. Patients were said to improve greatly during pregnancy and, on the assumption that the disease was due to a disturbance of the internal secretion, polyglandin had been prescribed. Dr. Farquhar Buzzard reported that these patients had creatin in the urine, and that this substance was absent from the muscles. Dr. Buzzard also

<sup>1</sup> For a full report of this case, vide *Guy's Hospital Gazette*, October 28, 1922, p. 445.

## 18 Symonds: *Myasthenia Gravis*; Tilley: *Myotonia Atrophica*

published in *Brain*, 1905,<sup>1</sup> the reports of five post-mortem examinations in cases of myasthenia gravis. He asked whether Dr. Symonds had tried polyglandin, and with what result.

Mr. ARCHER RYLAND pointed out that there was a marked reduction of sensibility of the soft palate in this case. When this sign was present, he was always suspicious of a possible "hysterical" condition. He asked Dr. Symonds whether, in his opinion, this phenomenon—as an indication of "hysteria"—had been overrated.

Mr. SYDNEY SCOTT said he had seen several of these cases through his association with the National Hospital at Queen Square, but when he saw them the diagnosis had been already made, and it became a matter only of laryngeal examination. He had noticed the ptosis, the weak muscular movements of the palate and tongue, as well as the laxity of the vocal cords. He suggested that possibly the apparently reduced sensitiveness of the palate, present in these cases, was comparable to the apparently sluggish corneal reflex in cases of facial paresis; and he asked if Dr. Symonds agreed that this was so.

Dr. C. P. SYMONDS (in reply) said the prognosis of the condition was less serious than was generally supposed. The danger was in the fatigue involving the respiratory muscles and so causing death from respiratory failure. It some cases artificial respiration had been kept up for many hours, and the patients lived for a year afterwards. Spontaneous remissions were characteristic of the disease. The patient in one of the five cases he had seen died six months after the onset of the disease. The present patient seemed to have had some symptoms for eighteen years, based on the ptosis. It was difficult to make a prognosis in any given case. These patients had marked freedom from the symptoms during pregnancy. One patient in hospital was transfused with blood from a pregnant woman, but without benefit. He was now trying large doses of corpus luteum, and the patient seemed better. The creatin findings fitted in with the low muscular metabolism; further observations were required before it could be known that abnormal creatin output had anything to do with causation in this disease. He did not quite agree with Mr. Scott's explanation of the apparent anaesthesia of the palate. Many of the patients complained of sensory symptoms; in one case the symptoms began with a loss of taste, and that was followed by anaesthesia in the tongue. The present patient herself discovered the anaesthesia of the palate accidentally. He did not regard anaesthesia of the pharynx as a valuable sign in hysteria. The so-called stigmata of hysteria could be produced in most people by suggestion.

### Case of *Myotonia Atrophica* with Implication of Left Crico-arytænoid Muscle.

By HERBERT TILLEY, F.R.C.S.

PATIENT, a male, aged 26. History: For five years has complained of increasing muscular weakness which is aggravated by exertion. This was first noticed when, as a soldier, marching made his legs "flop about" and he had great difficulty in rifle drill.

Past illnesses: Malaria, sand-fly fever, dysentery, influenza, and "dry pleurisy." No history of syphilis.

Family history: No similar case known to patient.

Present state: Patient is a thin, under-developed man, of dull, apathetic appearance. Voice high-pitched and weak. Speech is rather indistinct and

<sup>1</sup> Buzzard, E. Farquhar, "The Clinical History and Post-mortem Examination of Five Cases of Myasthenia Gravis," *Brain*, 1905, xxviii, p. 438.

words tend to run together. He gives the impression of being weak and stiff in his movements.

**Cranial nerves:** Lateral eye-movements deficient; ptosis each side; weakness of all facial muscles due to wasting and most marked on left side. Sagging of lower jaw; slight difficulty of swallowing; sterno-mastoids very weak, especially left side.

**Motor system:** Weakness of all facial muscles; marked ptosis each side. Delay in relaxation of face muscles after percussion. Tongue not wasted but relaxation prolonged. Sterno-mastoids both wasted and clavicular portions have practically disappeared. Slight general wasting of both upper limbs, most marked in the inner flexors of forearms. Wasting and extreme weakness of vasti externi, peroneal and anterior tibial groups of muscles and hence the tendency to inversion and dropping of feet. The reflexes of the upper limb are very sluggish and are absent at the ankle-joint. Rombergism well marked.

**Larynx:** In phonation the left vocal cord is motionless in the middle line. (The patient is under the care of Dr. T. R. Elliott, Medical Unit, University College Hospital, who has kindly allowed the patient to be shown and placed the above notes at the disposal of the exhibitor.)

### **Case of Multiple Foci of Growth in the Palate and Tonsil.**

By W. M. MOLLISON, M.Ch.

PATIENT, a male, aged 64, noticed patches of ulceration on the palate about ten months ago; under potassium iodide (20 gr. t.d.) improvement, but some patches remain. About five or six months ago glands appeared on the right side of the neck but have disappeared; for some weeks past a swelling at the angle of the jaw on the left side has been so painful as to keep the patient awake at night; it is also hard and tender. At the upper pole of the left tonsil is a hard nodule involving the anterior pillar. On the hard palate are three or four small raised patches, while on the most anterior part is a larger area of ulceration. Microscopical examination shows one of the patches to be carcinoma.

#### **DISCUSSION.**

MR. CHARLES A. PARKER (President) asked from which growth a portion had been taken for examination. He thought the small growth on the palate might be inflammatory, due to an ill-fitting dental plate, and quite independent of the growth in the tonsil, which was evidently malignant.

MR. STUART-LOW said he had not previously seen a case of this kind in which such multiple ulceration had occurred. The growths were hard and tender on palpation, and did not appear to be of an inflammatory nature, particularly on the right tonsil. He regarded these discrete deposits as breaking down gummata which had become malignant. This patient presented some of the conditions which in his (Mr. Stuart-Low's) opinion led up to malignant disease of the throat—e.g., excessive smoking, swallowing of very hot food, septic mouth, with marked acidity of the fluids in the mouth, taking a large quantity of salt with food, and syphilis. In his (the speaker's) opinion nothing could be done for the patient except alleviation of the symptoms, and he advised mucin as a mouth-wash and spray to counteract the acidity of the mouth and pharynx.

Mr. MOLLISON (in reply) said he would report later on the further pathological findings.

*Later report* (January 8, 1923): Section of a second nodule removed did not show carcinomatous tissue.

### **Congenital Webbing of the Larynx.**

By G. W. DAWSON, F.R.C.S.I.

FEMALE, aged 21. Complains of hoarseness for many years. No history of any laryngeal affection. A crescentic web with concavity looking backwards is seen in the anterior portion of the larynx stretching between the vocal cords.

### **A Woman whose Larynx can be examined by the Direct Method with the aid of a Tongue Depressor only.**

By G. W. DAWSON, F.R.C.S.I.

### **Skiagram showing a Paper Fastener in Left Bronchus of a Child.**

By HERBERT TILLEY, F.R.C.S.

THIS foreign body which had been impacted for twenty-one months was successfully removed by peroral endoscopy.

(Fuller notes of this case may be found in the *British Medical Journal*, November 18, 1922, p. 973.)

### **Microscopic Section of a Benign, Pedunculated Tumour of the Left Tonsil.**

By HERBERT TILLEY, F.R.C.S.

A SMALL ovoid tumour attached by a thin pedicle to the upper pole of the left tonsil, removed from a young adult. It measured  $\frac{1}{2}$  in. in length, and  $\frac{1}{4}$  in. in breadth. Microscopically it proved to consist of normal tonsil tissue. (H. G. Butterfield.)

### **Case of Chronic Empyema of the Antrum ; Canfield's Operation ; Recovery.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a male, aged 32, complained of fetid purulent discharge from the left side of the nose, of four years' duration, accompanied by left frontal headache. Transillumination showed opacity of the left antrum, and puncture gave vent to a quantity of fetid pus. The middle turbinal was enlarged, and the

anterior part of it was removed so as to free the infundibulum and the orifice of the antrum. This was followed by relief to the headache, but repeated punctures showed no diminution of the amount of pus. I operated by Canfield's method, and in a few days the purulent discharge had completely disappeared. A small mass of granulations developed on the anterior margin of the opening, which interfered with the introduction of a cannula, but after this was removed there was no further difficulty.

Sir JAMES DUNDAS-GRANT said he found that after Canfield's operation the cavities cleared up much quicker than by any other method. He had shown a number of these cases before the Section, and had described the operation in detail.<sup>1</sup>

### Case for Diagnosis.

By H. BELL TAWSE, F.R.C.S.

PATIENT, a female, aged 40. Slightly to the right of the middle line of the neck, lying over the upper part of the right ala of the thyroid cartilage and adjacent part of the thyro-hyoid membrane, there stretches up to the submental region a cystic swelling of the size of a pigeon's egg. It is painless, varies in size, and appeared in June of this year. On the anterior surface of the epiglottis on the right side and filling up the vallecula is a rounded, smooth, semi-translucent swelling with blood-vessels coursing over it. It appears to elevate the ventricular band and to obscure the vocal cord. It greatly impedes the movements of the right side of the larynx. The patient's voice is somewhat impaired, but otherwise she is well.

Opinions are asked as to diagnosis and treatment.

### DISCUSSION.

Mr. A. J. M. WRIGHT thought the swelling was a thyro-glossal cyst. In 1913, he showed a cyst he removed from a patient, and last week he operated on a similar case. These cysts were easily removed by dividing the body of the hyoid and removing the cyst without opening the mucous membrane.

Mr. NORMAN PATTERSON said the condition had evidently somewhat altered since the description was written.

Mr. H. BELL TAWSE (in reply) said he considered the swelling was a dermoid, and he agreed there had been a recent alteration in the appearance since the notes of the case were submitted. A fortnight ago the cyst was four times its present size, and the right vocal cord could not be seen. He had never seen a case in which there was so much extension upwards and to one side. It might have originated as a hyoid bursa, which had become cystic and extended up beyond the thyro-hyoid membrane to the region of the vallecula.

### Thyroid Tumour from Base of Tongue.

By H. BELL TAWSE, F.R.C.S.

PATIENT, a female, aged 52, had growth removed from throat over twenty years ago, said to be sarcoma; again one year later when it was said to be benign. Further operation on throat two years later—no history of result.

<sup>1</sup> *Proceedings*, 1921, xiv (Sect. Laryng.), pp. 41-42.



## 22 Tawse: *Thyroid Tumour; Submaxillary Gland*

No more trouble till last Christmas when she felt a "lump" in throat which gradually caused some difficulty in swallowing and, later on, some obstruction to breathing.

Seen by me last July. On depressing the tongue a brownish foul-smelling mass was seen protruding from the epiglottic region. Palpation by the finger showed it to be a soft, rounded tumour, bleeding freely on touch, and covered with stinking crusts. It seemed firmly adherent to the epiglottis, the tip of which was free from the growth. No dyspnoea except on exertion. A few anterior cervical glands enlarged on both sides. Three pieces removed—free bleeding. Pathologist's report: "Tissue entirely necrotic with the exception of one part which shows cavernous tissue." Wassermann test negative. Two larger pieces removed—bleeding so profuse as to cause anaemia. Pathologist's report: "Hæmangioma of the capillary type—most of mass necrotic."

August 28, 1922; Tracheotomy.—Subhyoid pharyngotomy gave insufficient room for removal of the growth and a transhyoid incision was made and the hyoid divided; growth appeared to be adherent to the epiglottis which was cut through and removed along with the mass which shelled out from the base of the tongue. No hæmorrhage. Extremities of incisions drawn together with deep placed silkworm gut, centre left open and drained.

August 29, 1922: Tracheotomy tube removed—can swallow fluids.

August 31, 1922: Skin red, swollen and inflamed; stitches removed: entire wound sloughing. Temperature normal; being fed by mouth or rectum.

September 2, 1922: Wounds cleaning; can swallow well, but some escapes from the wound.

September 27, 1922: Able to go home; wound closing rapidly.

October 10, 1922: Wound completely healed; patient very well.

Pathologist's report: "Sections show structure of so-called foetal adenoma of thyroid. A few acini contain normal staining colloid. Periphery of growth shows dense vascular supply and on the free surface it is entirely necrotic."

No thyroid gland palpable in the neck.

Microscopic section shown.

Mr. W. M. MOLLISON said Mr. Steward had recently reminded him of a case of thyroid tumour at the base of the tongue which he had removed. It was the only piece of that tissue present in the patient's body and its removal had necessitated thyroid extract being constantly taken by the patient ever since, and acute myxœdema followed.

### Submaxillary Gland with Calculi.

By H. BELL TAWSE, F.R.C.S.

X-RAY plates before operation, and of the gland after operation.

**Nasal Stenosis, mainly Subjective, in a Case of Parkinson's Disease.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D., and C. C. WORSTER-DROUGHT, M.D.

PATIENT, a male, aged 40, with the expressionless, "mask-like" face of paralysis agitans, but without shaking, was referred by Dr. Worster-Drought complaining of nasal obstruction. The turbinated bodies were somewhat hypertrophied, but not sufficiently so to produce the amount of obstruction of which the patient was conscious. The sensibility of the nasal mucous membrane was found to be lowered to a very great degree so that patient did not perceive the air passing through the nose, and consequently felt as if it did not do so—"subjective nasal stenosis."

DISCUSSION.

Mr. O'MALLEY said the patient had told him that he had difficulty in breathing when he lay down at night, or when he was in a warm place. This difficulty of breathing was accounted for by the hypertrophy of the turbinates present in the case, apart from any supposed loss of physiological function.

Dr. JOBSON HORNE said it seemed to be an ordinary straightforward case of an enlarged middle turbinal, which required reducing.

Dr. W. H. KELSON, referring to the nomenclature of diseases, considered that the better known term "paralysis agitans" was preferable to "Parkinson's disease."

Sir JAMES DUNDAS-GRANT (in reply) said that at first he described the case as "paralysis agitans," but Dr. Worster-Drought termed it "Parkinson's disease" (synonym). The case showed the physiognomy of this disease, though the patient was rather younger than usual.

**Case of Chronic Laryngitis of Long Standing.**

By C. A. S. RIDOUT, M.S.

PATIENT, a male, aged 50, was first seen in April, 1921, complaining of hoarseness of four to five years' duration. He gave a history of syphilis many years ago. Wassermann test negative. By occupation he is an instructor in physical exercises, &c. On examination of the larynx the right vocal cord showed destruction in middle one-third by old, now healed, ulceration, with a rounded prominence anteriorly. The left vocal cord showed considerable destruction and irregularity, a protuberance in the middle one-third seeming to fit into the depression in the right cord. Ventricular bands thickened. Movements good. During the past eighteen months patient has had potassium iodide at intervals, inhalations of menthol, astringent sprays and mercurial inunction into the thyroid alæ. A small portion of tissue removed with the forceps showed no malignancy. The condition has changed of late, the voice is still hoarse, movements of the vocal cords remain good, the depression in the right vocal cord seems to have filled up. Both cords, however, are thickened and irregular and the left cord shows a sessile raised whitish patch in its middle one-third with an oblique cleft separating it from a pinkish swollen posterior one-third.

Treatment has recently been discontinued.

The opinion of the Section is desired: (a) As to the nature of the condition, especially that of the left vocal cord; (b) as to future treatment; (c) as to prognosis.

#### DISCUSSION.

Mr. CHARLES A. PARKER (President) said he had seen a similar condition in patients who had had syphilis and used the voice excessively, in one instance, in a butcher, who cried his meat outside his shop. He considered the condition was due to chronic laryngitis profoundly influenced by the fact that the patients had had syphilis, though the lesion itself was not syphilitic. In his cases treatment had been of little use and he thought the prognosis as regarded the voice was unfavourable.

Dr. JOBSON HORNE agreed with the President, and remarked that it was the kind of case which suggested epithelioma. The clinical history, the symmetry, and the absence of gland involvement, however, were against epithelioma. There was no ulceration in the larynx, merely thickening of the vocal cords and epithelium. He regarded it as a case of pachydermia syphilitica. He had seen at autopsy a larynx in such a case and had microscoped it; and it showed a considerable heaping up and thickening of squamous epithelium. There was a tendency for the condition to spread below the larynx, thereby placing the patient in difficulties. He did not think the condition would improve, except temporarily. He knew of one case in which tracheotomy became necessary. The less the patient (now exhibited) used his voice, the better, and he should take very little alcohol. The only drug which gave any hope was iodide of potassium. About eight years ago Mr. Tilley showed a woman with such a condition, which was thought to be malignant. He had examined the larynx in that case and would exhibit the specimen at a later meeting. It illustrated dovetailing of the cords as the disease progressed.

Mr. TILLEY spoke of a similar case which he had recorded in the *Proceedings* in 1916,<sup>1</sup> under the title of "Leucoplakia of the Vocal Cords," in which there were lenticular leucoplakic patches on the upper surface of the anterior third of each vocal cord. Antisyphilitic remedies proved futile, and the condition remained the same for about two years, when patient was lost sight of. In discussion one member of the Section suggested that the lesions were early tubercular, but no other signs of tubercle were present. The patient referred to by Dr. Horne insisted on leaving hospital, and two days later she suddenly expired. At autopsy a hobnailed liver was found, in addition to extensive interarytenoid hyperplasia referred to by Dr. Horne. Possibly this was only local evidence of a general fibrosis of different organs.

Mr. E. D. D. DAVIS said he had seen two cases of the same kind. The first was that of a salesman who talked, drank, and smoked too much. The ventricular bands were oedematous and obscured a view of the vocal cords. Iodide of potassium was prescribed in large doses without any improvement. The second patient was an engine-driver, on whom he (Mr. Davis) had performed laryngo-fissure, as the condition was thought to be an epithelioma. It proved to be tubercular, and the sputum was found to contain tubercle bacilli. Patient was still alive and well.

Dr. WILLIAM HILL said this case showed an unusual leucoplakic pachydermia, with an irregular surface, and indentation of the right cord. He thought the application of the galvano-cautery would reduce the tumefaction. [Dr. JOBSON HORNE: The galvano-cautery would aggravate the pachydermia.]

Dr. BROWN KELLY suggested that the condition on the left cord was epithelioma. The right cord presented healed ulceration which might have been syphilitic.

Mr. RIDOUT (in reply) said he regarded it as a pre-cancerous condition. He had been in doubt whether he ought to watch the case or take immediate active measures. After hearing the discussion he had decided to watch the case, but he thought that it would soon be necessary to deal with it surgically.

<sup>1</sup> *Proceedings*, 1916, ix (Sect. Laryng.), p. 67.

## Case of Pedunculated Angeioma (Bleeding Polypus) of the Inferior Turbinal.

By SOMERVILLE HASTINGS, M.S.

PATIENT, a female, aged 24, complains of slight nasal obstruction relieved by blowing out a piece of fibrinous exudate and accompanied by hæmorrhage every evening, together with a feeling of fullness in the nose. She gives two months' history of these symptoms. A pedunculated growth will be seen growing from the upper part of the right inferior turbinal, about the size of a pea. It bleeds very easily when touched. The case would seem to resemble a similar one shown by the writer on March 4, 1910.<sup>1</sup>

### DISCUSSION.

Dr. W. H. KELSON said that this tumour was formerly described as bleeding polypus of the septum, on account of its supposed limitation to that locality. It had more recently been shown that such tumours sometimes originated from other parts of the nose, e.g., the inferior turbinals. Sir StClair Thomson, in the latest edition of his textbook on "Diseases of the Nose and Throat," had described these tumours under the title of "bleeding polypus of the nose."

Mr. SOMERVILLE HASTINGS (in reply) said he would remove the tumour, and show the sections at a later meeting.

## Cyst of Uvula.

By T. JEFFERSON FAULDER, F.R.C.S.

PATIENT, a child, aged 10 months. The uvula was completely involved by a translucent cyst, except at the tip. Scanty fibres and minute vessels were spread over the surface. The mother stated that it had been present since birth. It had produced no symptoms, but was getting larger. Removed by cold snare.

## Two Cases of Pulmonary Tuberculosis with Laryngeal Symptoms.

By PHILIP FRANKLIN, F.R.C.S.

*Case I.*—Patient, a male, aged 49, who has suffered from pulmonary tuberculosis since 1918, and has undergone seven months' sanatorium treatment. Sputum and X-ray positive. Larynx shows swelling of the left aryteno-epiglottidean with ulceration of the left arytenoid cartilage. No pain. This case shows characteristics similar to the next, yet the larynx is undoubtedly tuberculous.

*Case II.*—Patient, a male, aged 40, who had an attack of hæmoptysis, February, 1922, and since then he has been under treatment for pulmonary tuberculosis. Three weeks ago he developed a husky voice. X-ray confirms

<sup>1</sup> *Proceedings* 1910, iii (Sect. Laryng.), pp. 102-106. Microscopic sections were also published.

the physical signs of a cavity at the right apex; sputum contains tubercle bacilli. No pain. Larynx shows ulceration of the right arytaenoid. When first examined two weeks ago, the ulcerated area gave the impression that a growth was present. The Wassermann reaction is positive. After a week's treatment with potassium iodide the larynx rapidly improved. This case is of special interest in view of the syphilitic nature of the larynx, associated with advanced pulmonary tuberculosis.

#### DISCUSSION.

MR. CHARLES A. PARKER (President) said he thought one of the cases was typical tubercle of the larynx. If the other had been tuberculous there was now fibrosis, and activity had ceased.

MR. FRANKLIN (in reply) said that he brought the cases forward because of points of similarity between them. The case referred to by the President as showing fibrosis had had a definite ulceration a few weeks ago, and a three weeks' history of hoarseness. When first seen it did not appear to be a typical case of tuberculosis. A Wassermann reaction proved to be positive. The condition was now clearing up under iodide of potassium. The other showed a similar condition of the larynx, yet was definitely tuberculous.

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THE following cases, together with the discussion, have been deferred for publication until further investigations or completed reports have been submitted:—

- (1) H. BELL TAWSE, F.R.C.S.: "Ulceration of the Left Tonsil—for Diagnosis." This case was previously shown at the meeting of the Section, held November 3, 1922, and was deferred for further investigation. The exhibitor now reports a large ulcer on the anterior pillar of the fauces, spreading on to the palate.
- (2) W. M. MOLLISON, M.Ch.: "Laryngeal Case—for Diagnosis."
- (3) T. JEFFERSON FAULDER, F.R.C.S.: "Papillomatous Growth of Larynx—for Diagnosis."

## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### Case of Œdema of the Septum in association with Nasal Polypi.

By A. J. WRIGHT, F.R.C.S.

PATIENT, a male, aged 40. Nasal discharge and obstruction for ten years. Polypi removed on many occasions, and intra-nasal operations on antra and ethmoidal cells. In April, 1920, limited submucous resection. Since then, slowly increasing swelling of septum. Suggestions as to treatment are invited. Would removal of the septum be justifiable and helpful?

#### DISCUSSION.

Dr. WILLIAM HILL suggested that a piece be cut out, extending, if necessary, through the whole thickness, to ascertain whether it was lymphangitis of exceptional form, or a solid pseudo-œdema in the pharynx.

Sir WILLIAM MILLIGAN said he understood that this swelling had been punctured, but no fluid was present. The condition gave him the impression of being a very low form of perichondrial infection, dating from the operation. He did not advise removal of any portion of the septum. He suggested that diathermy with a very fine needle would not only sterilize it, but also cause sufficient cicatricial contraction to bring the two sides of the potential cavity together, and make a fairly rigid septum.

Mr. W. STUART-LOW said he had had a similar case, but less severe. The patient was employed at dusty work, and the present patient similarly worked in grain dust, which was sometimes very irritating. He (the speaker) preferred oily preparations to lotions, since they supplied better protection from the dust. In the present case a discharge was seen in the upper part of the nasal cavities, and he asked whether this discharge contained streptococci, as these organisms were very irritating to the mucous membrane and might help to explain the condition. In his own case he first improved the patients' general health, and employed oily applications. Later he incised the swelling freely and packed the nasal cavity with strips of gauze soaked in liquid iodex, and under this treatment the swelling rapidly subsided and did not recur.

Dr. SYME agreed that it was a case of septic œdema due to disease of the ethmoids and antral cavities, which were discharging freely. He advised opening the maxillary antra by the canine fossa route, and removal of the diseased ethmoid plate.

Dr. DAN MCKENZIE remarked that he had had a number of similar cases, and had found a difficulty in explaining why the œdema should be most strongly marked at the

bony part of the septum; the cartilaginous part being not so much involved. It was always associated with sepsis of the ethmoid, and in the cases he had seen the condition improved to a moderate extent after the ethmoids had been thoroughly removed.

Dr. SMURTHWAITE said he had had a similar experience after septum resection. He incised the swelling and found a big cavity with septic serum inside, which he curetted, this being followed by the treatment mentioned by Mr. Stuart-Low, and in a week the surfaces united. Union did not occur at first, as there was a cavity filled with blood, which became septic. He advised this treatment, following removal of unhealthy ethmoids.

Mr. M. VLASTO said that palpation with the little fingers in each nostril showed that the swelling was not so marked as it appeared to be on inspection. The fingers were arrested by a distinct vomerine ridge, the removal of which by a more complete resection would probably get rid of some of the obstruction.

Mr. SOMERVILLE HASTINGS said he had watched two cases similar to the one now shown, for at least seven years: one in which the ethmoids had been thoroughly removed, and there was no discharge present, yet the swelling continued. The other case—which at first sight suggested a nasal polypus—the President had seen with him at the Middlesex Hospital some years ago. He had received various suggestions for treatment such as cutting out pieces, burning with the cautery, &c.; all these he had tried but with very little benefit. He was, therefore, glad to hear suggestions as to the treatment of this condition.

Dr. BROWN KELLY said he had had two similar cases following submucous resection, with marked soft swelling on both sides of the septum causing antero-superior bulging. Both patients had suffered from vasomotor rhinitis, and to this he attributed the swellings. It was conceivable that after the submucous resection and before complete healing had occurred, the frequently recurring swelling and œdema of the tissues, especially of the erectile tissue in the region of the septal tubercles, would force the flaps apart and keep them apart sufficiently long to allow of loose connective tissue developing between. On interrogating the present patient symptoms of vasomotor rhinitis were found to be present. The presence of pus also raised an interesting point. He had had several cases in which vasomotor rhinitis or asthma was associated with accessory sinus disease, and when the suppuration was cured, the asthmatic and vasomotor symptoms passed off. In these cases there was, almost surely, sensitization of the patient to bacterial proteins.

Mr. J. F. O'MALLEY said that in 1911 he exhibited before the Section an identical case though not so extensive; in which there was double ethmoid sepsis with polypi, and much bilateral œdema of the septum which anyone could mistake for polypoid masses protruding into the passages. In that case he removed the ethmoid cells, but no improvement in the œdema followed. He then resected the septum, with likewise no improvement. He next excised a wedge without causing perforation. And this made only a slight alteration in the condition. He concluded it was due to some lymphatic obstruction, and so long as there was a mass of tissue there, with the lymphatic outlet obstructed, a certain amount of œdema must occur. Sir William Milligan's suggestion might help to relieve the tendency to the œdema, by fixing the tissues and reducing their size.

Sir STCLAIR THOMSON suggested that as this swelling had been punctured and no fluid was found, the title should be altered to "pseudo-œdema," since the present title was misleading. As to the nature of the tissue, he referred Mr. Wright to some work done by Dr. Pegler, many years ago, and shown to the Section, in which this condition was present without polypi, and without a septum resection having been performed.



Dr. Pegler's slides showed the swelling to be a lymphoma. The speaker had seen a fair number of these cases with and without sinus trouble and septum resection. One of them was in the early days of septum resection, when he performed the operation for hay fever, but it left the patient in a worse state than before, because formerly there was only a temporary turgescence of the septum, whereas a permanent condition followed. His own experience confirmed the opinion of Mr. Somerville Hastings, and that of Dr. Brown Kelly, that this condition resulted from a neurosis, probably sensitized by infection from the ethmoid and other sinuses.

Mr. CHARLES A. PARKER (President) said the discussion had shown these conditions were not uncommon; he could himself recall several cases, and he agreed that no method of treatment seemed to be of much service. He had tried removal of the swelling, but recurrence occurred. He agreed with Sir William Milligan's view that there was probably an underlying perichondritis or periosteitis, which led to a difficulty in removing the whole disease and hence to recurrence.

Mr. A. J. WRIGHT (in reply) said he thought there was no cavity present, but the swelling could be emptied by slow pressure, a fact which was against it being a post-hæmorrhagic or post-suppurative collection of fluid. It was in the tissues of the septum. Having watched this slowly develop for two years he thought the patient would be more comfortable by removal of the whole swollen septum, but he had not had the courage to do this, and no one in the discussion urged it. The patient had ethmoidal suppuration and nasal obstruction, and if the ethmoid cells could be sufficiently removed the condition of the septum might improve. He had not tried diathermy, but on two occasions he had inserted a galvano-cautery point into the swelling, making a submucous linear cauterization, which was followed by further swelling. Therefore he was somewhat doubtful as to the result which would follow diathermy. In this class of case vasomotor rhinitis was not an entity, in his view; he did not know where the pure vasomotor condition ended and where the suppurative began. Patients suffering from ethmoidal suppuration seemed frequently to have attacks of sneezing, with a watery discharge; but he had hesitated to call the attacks vasomotor rhinitis in the presence of an obvious gross infection.

## **Bismuth and Glycerine Gauze.**

By Sir STCLAIR THOMSON, M.D.

THE strips of gauze are laid evenly over the mucous surfaces of the outer and inner walls and kept in position with others between them. About three or four strips are used in each nasal cavity, which is not tightly plugged. This allows of some drainage. When removed at the end of thirty-six to forty-eight hours the absence of reaction and the quiet condition of the mucous surface is remarkable.

The gauze is prepared as follows: The gauze, which is of fine mesh, is cut 1 in. wide and 3 in. long, removing all lint and threads. Soak the gauze in equal parts of glycerine and water, dry with towel all excess moisture. Impregnate by rubbing on sufficient bismuth subcarbonate to cover the gauze, but not enough to have an excess quantity which would fall off. Put up in packages of two dozen and sterilize in steam sterilizer.

Since I began to do submucous resection of the septum in 1901, I have tried all the various packings—cotton-wool, gauze, Lister's protective, rubber-sponge, &c. I have even tried to do without any packing, but have returned to its use.

In America, in the summer of 1919, I saw the good results of bismuth gauze

in Dr. Coakley's clinic. Previously I had made a trial of dry bismuth gauze without marked results, but this bismuth and glycerine gauze has proved remarkably satisfactory.

#### DISCUSSION.

Mr. H. J. BANKS-DAVIS reminded members of the possibility of nose-splints being swallowed by patients unless they fitted tightly. Some which were stated to have been "sneezed out" by the patient were eventually passed *per rectum*, having been sucked into the nasopharynx and swallowed with the nasal hypersecretions.

Mr. W. G. HOWARTH said that he could testify to the value of these plugs. He had used similar plugs for the last ten years since he saw Dr. Coakley, of New York, use them in his clinic. He however preferred the subgallate of bismuth to the subcarbonate.

Dr. BROWN KELLY said that for many years he had used plugs of bismuth subnitrate, which were introduced by Dr. J. L. Howie. These were easily removed, and could, if necessary, be left in the nose for several days.

Sir STCLAIR THOMSON (in reply) added that the hygroscopic action of the glycerine kept the turbinates from getting congested, and that the bismuth was very soothing to the mucous membrane. It was the most satisfactory preparation he had ever tried.

### Epithelioma of Nasal Septum, Floor of both Nostrils, Alveolar Surface Upper Jaw, and Left Side Lower Jaw.

By ANDREW WYLIE, M.D.

PATIENT, a man, aged 34, painter, complained of an obstruction in his left nostril for four weeks. On examination a hæmorrhagic spongy growth, bleeding profusely if touched, is seen on both sides of septum, and has caused a perforation. There is the same condition on the floor of both nostrils, the alveolar surface of upper jaw and the left side of lower jaw. The disease is spreading rapidly. On transillumination distinct dullness on the right side. No enlargement of the spleen. Pieces removed from the septum and jaw and sent to pathologist. Dr. Scott Williamson reports: "A very malignant epithelioma." Wassermann negative.

Dr. Salisbury Sharpe examined the blood and reports: "Hæmoglobin" about 77 per cent.; red cells, 2,308,000; white cells, 10,360; slight leucocytosis."

The disease is spreading, and the exhibitor wishes opinion on treatment. He intends to get some teeth removed and apply diathermy, if possible, to the nose. A microscopical slide is shown.

[The case was seen at the special meeting held on January 5.]

#### DISCUSSION.

Mr. H. J. BANKS-DAVIS said it seemed almost impossible that the condition could be an epithelioma, as it had started at so many points. He understood from Dr. Wylie that the patient was at present almost at death's door, and he did not think epithelioma could make a man so ill in so short a time. With regard to the microscopical section it seemed to him to consist of small round cells and inflammatory tissue: he did not observe indications of so typical an epithelioma as it was said to be.

Dr. WYLIE (in reply) stated that the patient was very ill and was not able to swallow food, and that the disease was spreading. He hoped to get further specimens of the diseased tissue for examination.

**Laryngeal Case for Diagnosis.**

By H. SMURTHWAITE, M.D.

A. R., MALE, aged 45. Had large papillomatous mass removed from larynx in July, 1917, by Dr. Brady, of Sydney. History of twelve years' huskiness previously. Now slight hoarseness, though no worse than after operation. Chief trouble, intermittent pain in right side of throat below tonsil. Larynx: Small outgrowth on left cord just above centre—no lagging, no ulceration; cord moves freely. Suggestions as to nature of growth and as to treatment, if any, are invited.

**DISCUSSION.**

Dr. DAN MCKENZIE said he saw this case immediately after the original growth was removed five years ago by Dr. Brady of Sydney, and the condition of the larynx had not changed much, except that the masses had increased in size and become more lobulated, and also there was now to be seen a similar patch, which looked like pachydermia, of the right cord. The slow development of the case, its quiet appearance, and the fact that there was also a similar patch on the other cord, led to the hope that it was not malignant, but probably pachydermia.

Mr. CYRIL HORSFORD said he thought the patch which had developed on the right cord was of similar structure to that on the left, and he regarded it as papillomatous. The pain on the right side of the throat he believed was due to retained pus in the right tonsil.

Sir JAMES DUNDAS-GRANT thought it advisable to remove a portion for microscopical examination. It was unlikely to be a malignant condition. If the growth interfered with the voice more than it did, the indication for what he suggested would be even stronger.

Dr. W. H. KELSON said this larynx looked like what one would expect in a patient who had had a large papillomatous mass removed a few years before, i.e., exhibiting stumps, apparently non-malignant.

Mr. J. F. O'MALLEY said that the patient complained of pain at one particular spot on the right side of the neck, and on examination some secretion was seen to be held up in the pyriform fossa on that side. If the patient was asked to press outside on the spot about which he complained, it disturbed the position in which the fluid was held up, while when he relaxed his finger it was seen to recede. He could not see evidence of growth beneath the secretion in the pyriform fossa. He agreed there was general thickening of the cord.

Mr. ARCHER RYLAND was of the opinion that the small fleshy outgrowth of the left cord was producing an irritative lesion at the corresponding point on the right cord. He saw no reason why the small tumour should not be removed, and thought this would probably result in improvement in the voice.

Mr. H. SMURTHWAITE (in reply) said that the man had complained a good deal about the right side of the pharynx from time to time, but he (the speaker) could find nothing wrong there. He probably had a small concretion in the tonsillar fossa on the right side, causing the pain. His desire had been to ascertain whether it was advisable to remove the left cord.

**Operative Procedures in the Treatment of Stenosis of the Larynx caused by Bilateral Paralysis of the Abductor Muscles, with Special Reference to a New Method by means of which it is suggested that the Airway may be Permanently Enlarged, and the Patient Decannulated.**

(ABSTRACT.)

By IRWIN MOORE, M.Ch.

(I) INTRODUCTION.

IN this communication I wish to mention the various operative procedures which have been suggested in the past for the permanent relief of laryngeal stenosis caused by bilateral abductor paralysis, and to bring to notice some preliminary investigations which I have been carrying out.

Professor Hobday recently suggested the possibility of permanently re-opening the airway in cases of double abductor paralysis by ventriculectomy as performed in the horse for unilateral recurrent paralysis (roaring). This suggestion has set one thinking that perhaps we are lacking in our endeavours—in not trying to do something more than tracheotomy for those unfortunate people who are suffering from this distressing condition.

Tracheotomy is only a life-saving device, and leaves the patient in a position of prolonged dependence on a breathing cannula. Have we, then, at the present day, any satisfactory method by means of which, in cases of double abductor paralysis, the airway may be re-opened, and the respiration through the natural passages restored?

We know that the abductor and adductor muscles of the vocal cords are supplied by the inferior laryngeal nerves, and that any destructive lesion of the nerve or its centre in the medulla produces its effect by paralyzing first the abductor muscles, and finally the adductors.

In double abductor paralysis it is the unopposed action of the crico-arytenoideus lateralis, along with paralysis of the crico-arytenoideus posticus and the thyro-arytenoideus, which protrudes the cords into the airway of the larynx, and causes the stenosis. The cords become flaccid, and will not separate, and their edges flap up and down. The complete stage of paralysis of the recurrent nerves, i.e., the cords lying in the cadaveric position, usually allows of a sufficient airway, and prevents urgent dyspnoea, and the necessity of tracheotomy.

Amongst the various causes, bulbar lesions are the most common. In 90 per cent. of the cases they are tabetic in origin. Of fifty-three cases of abductor paralysis recorded between 1892 and 1898 only six had complete recurrent paralysis. Of the balance twenty-two had unilateral, and twenty-five bilateral, abductor paralysis. These statistics show the small chance of cases of double abductor ever reaching the stage of complete paralysis, i.e., with the cords in the cadaveric position.

(II) OPERATIVE TREATMENT.

It is said that any operative treatment for opening the airway is contra-indicated in the first six months, since occasionally spontaneous recovery may

occur. Chevalier Jackson thinks it better to wait for a year, for recovery after that time is probably impossible. After waiting so long, however, certain operative procedures are rendered useless owing to contraction and atrophy of the thyro-arytænoid muscle, or fixation of the crico-arytænoid joint.

(1) *Simple Division of the Recurrent Nerve.*

This has been carried out with the idea of producing the cadaveric position of the cords and so relieving the stenosis, but the results have been very disappointing. Though Chevalier Jackson has operated in one case followed by the cadaveric position of the cord, he agrees that the operation has been a failure. This failure is probably due to shortening of the adductors, which results from long contraction without antagonism, so that the vocal cord still keeps a median position.

(2) *Re-establishment of Nerve Continuity by Resection and Anastomosis.*

(a) Without transplantation. (b) With transplantation, e.g., the recurrent with the pneumogastric nerve. Its use is limited to recent cases of peripheral cervical lesions, e.g., following thyroidectomy.

(3) *Corpectomy, i.e., Excision of only the Cord itself.*

This has been carried out by (a) the indirect method with the laryngoscopic mirror, or with Kirstein's autoscope; (b) thyrofi ssure.

The results have not been successful on account of excessive granulations which follow the operation, necessitating re-insertion of the tracheotomy tube.

(4) *Arytænoidectomy.*

Since the arytænoids are the cartilages which move the vocal cords, it was natural that they should receive the attention of operators. Arytænoidectomy was first employed by veterinary surgeons in horses in 1834, and onwards to 1905 for the relief of laryngeal stridor ("roaring"), due to paralysis of the left vocal cord. Ivanoff in 1911 first performed this operation in man in a tracheotomized syphilitic patient whose cords were abducted. He carried out a unilateral arytænoidectomy, but after a time the cord became loosened, and respiration was impeded, this necessitating partial removal of the cord. The results were unsatisfactory, also, in a few other cases recorded.

(5) *Ventriculectomy or "Stripping" of the Lining Membrane of the Ventricle.*

In 1906, Williams (Cornell University, U.S.A.), finding that removal of the cords and of its muscles in the horse predisposed to recurrence of stenosis, originated *per se* excision of the lining membrane of the ventricle.

This operation consists in excision of the everted ventricular mucosa, putting out of action the adductor power of the vocal cords, which become attached by cicatricial contraction to the lateral wall of the larynx, in the position of forced abduction.

Since 1906 veterinary surgeons have found ventriculectomy a better operation than arytænoidectomy in cases of recurrent nerve paralysis.

Hobday in 1910 considerably improved this operation by reaching the larynx through the crico-thyroid membrane, and stripping both ventricles at the same time.

Sargnon and Toubert, collaborating in a paper published in 1914, suggested that ventriculectomy should yield the same favourable results in the human subject as in veterinary practice, and came to the conclusion that whilst it was easy to perform in the horse because the operator is able to introduce the end of his index finger into the ventricle, it was very difficult to perform in man, even on a cadaver, and that the only applicable method in the human subject was by scraping and curettage of the membrane.

Professor Hobday has recently suggested (1921) that ventriculectomy as performed in the horse might be applicable in man in cases of double abductor paralysis.

Monselles (Florence) in 1900 attempted to reproduce the condition of eversion of the ventricle artificially in thirty-three cases in the human subject after death, without success; in every case the mucosa instead of the mucous membrane was torn in attempting to draw out the lining membrane of the ventricles.

Vlasto (1921) has contrasted the anatomical difference between the ventricle and the sacculus in the horse and in man, pointing out that in the horse the ventricle and sacculus form one large cavity, extending downwards below the vocal cord and lining its outer surface; that its attachments are extremely loose, and can be easily stripped off in one piece, leaving raw surfaces opposed to one another, which adhere, and so cause a lateral retraction of the vocal cord. But in man the ventricle is very difficult to evert in the same manner. Therefore he does not think the operation is practicable or possible in the human subject.

Piersol has pointed out that in man the upper surface of the true cord slants only slightly downwards and outwards. The question whether the superior and outer surface of the cord, if stripped of its membrane, is of sufficient extent to adhere to the raw surface of the lateral wall of the ventricle, and so cause lateral retraction of the cord, is doubtful.

I have found, by operating on the cadaver, that the ventricle and sacculus cannot be everted and removed by the same method as in the horse, as described by Professor Hobday: in every case the forceps tore through the mucosa. It is possible, however, by dissection, to detach and remove the ventricular lining in one piece, if after complete detachment it is excised at its anterior attachment to the mouth of the sacculus, the latter being left *in situ*.

It is very difficult to detach the sacculus either along with the ventricle or even after removal of the ventricle, on account of the firm attachment of the suspensory ligament of Hilton, which slings up and supports the sacculus. Again, the mucous membrane covering the inner surface of the sacculus is very thin, and is so firmly adherent to the saccular wall, that it readily tears during attempts at separation. In only one out of eight attempts have I been able to remove the sacculus along with the ventricle.

If removal of the ventricular wall is ever attempted for treating abductor paralysis in man, it will probably be found advisable to remove only the ventricular lining, the sacculus being left undisturbed to carry on its normal function of mucous secretion. With regard to adhesion of the raw surface of the vocal cord to that of the lateral wall of the ventricle, I suggest that temporary fixation of the vocal cord to the lateral wall by means of a suture passed through the vocal cord and thyroid ala and tied externally, might encourage permanent adhesion and retraction.



(6) *Evisceration or Ablation of the Vocal Cord and the soft parts lining the Larynx.*

This operation may be carried out endoscopically or by thyro-fissure. Chevalier Jackson has had one successful endoscopic case. He states that he has found "evisceration" by thyro-fissure ideal in cases in which no other lesion is present beyond the actual bilateral paralysis, but considers that endoscopic evisceration is preferable. In two cases he operated on by the latter method the patients were permanently decannulated, and had a fairly loud voice. In both these cases all the soft tissue of the cord (and not simply the cord), including the subglottic tissue, was removed by dissection on both sides, leaving the perichondrium intact. Both patients were able to dispense with their tracheotomy tube.

(7) *Ventriculo-cordectomy.*

This operation, introduced by Chevalier Jackson, may be carried out either endoscopically or by thyro-fissure, and consists in excision of the vocal cord along with its supporting tissue forming the *floor of the ventricle*, by means of punch forceps. Chevalier Jackson has performed this operation endoscopically in eighteen cases under cocaine anaesthesia without any operative mortality. In eleven cases the operation failed to re-open the airway, on account of complications, e.g., cicatricial stenosis, and other means had to be employed. In the remaining seven cases, the patients were relieved of dyspnoea.

Molinie (Marseilles) in 1913 first considered the possibility of deviating the paralysed vocal cords laterally from the middle line, by displacing their anterior attachments inwards, i.e., antero-posteriorly. He first operated upon the cadaver by an incision through the midline of the thyroid cartilage without opening the cavity of the larynx, i.e., he did not incise the internal perichondrium. Two similar lateral incisions were next made on each side 5 mm. from the middle line incision, leaving two loose lateral pieces of cartilage (attached to perichondrium) which could be pressed inwards towards the centre of the larynx and kept in position by a steel band, thus diminishing the antero-posterior diameter of the laryngeal box, and causing deviation of the cords towards the lateral wall of the larynx. By this means (in the cadaver) an ovoid glottis was obtained. Molinie, thinking that the same results would follow in the living, performed a similar operation on a male patient, aged 25, suffering from bilateral abductor paralysis. The operation was a failure, and a tracheotomy tube had to be reinserted.

(8) *Cordopexy<sup>1</sup> or Antero-lateral Transplantation of the Vocal Cord.*

Wilfred Trotter recently (1922) suggested a very ingenious method of dealing with cases of abductor paralysis, by means of which he considered it might be possible to re-open the obstructed airway by transferring one or both of the paralysed cords from the middle line to the lateral wall of the larynx. He proposed that an incision should be made transversely across the middle of the thyroid cartilage, a retractor inserted, and the larynx opened so as to obtain a good view of the anterior insertion of the vocal cords. When they have been located the portion of cartilage to which they are attached could be

<sup>1</sup> *Postscript.*—To this operation I have—since the meeting—given the name "cordopexy."



separated from the thyroid ala by a circular incision, drawn forwards and carried laterally along the transverse incision through the thyroid ala. On removal of the retractor, the separated halves of the thyroid cartilage would come together and fix the cords in their new position. Much impressed with this idea, I obtained permission from Mr. Trotter to carry out investigations and ascertain its possibilities. Following out these suggestions on the cadaver it was found that the approach to the larynx by a transverse incision was not satisfactory, because it is impossible to locate accurately—from the exterior of the larynx—the anterior insertions of the vocal cords, and, avoid cutting into them.

Later the possibility presented itself of locating the point of origin of the vocal cords from the outside, and reaching them through a small incision in the middle line of the thyroid cartilage.

#### *Point of Origin of the Vocal Cords.*

Piersol, in his text-book of Anatomy, has pointed out that the true vocal cords arise, in both sexes, a little above the middle of a line from the bottom of the thyroid notch to the lower border of the thyroid cartilage. The anatomical studies of Taguchi showed that the average distance, in men, from the notch to the vocal cord was 8.5 mm. and from the lower border of the thyroid cartilage 10.5 mm. In women these distances were 6.5 and 8 mm. respectively, and the distance between the true vocal cords at their origin was 1.5 mm. in both sexes. Also, the false vocal cords arose about 2.5 mm. above the true ones, and were, on the average, 4 mm. apart from each other.

The apparent accuracy of these landmarks suggested to me the possibility of ascertaining the anterior insertion of the cords from the outside of the larynx.

After careful measurement, an incision a quarter of an inch in length was made through the centre of the middle line of the thyroid cartilage, and a bent probe inserted so as to locate the point of attachment of the cords. Next, a piece of cartilage (forming a triangle with the previous incision) was excised, to which (it was concluded) the anterior extremity of the cord was attached. From the apex of the triangle another incision was carried laterally through the thyroid ala for a quarter of an inch. The separated triangular piece of cartilage (with presumably the attached cord) was then drawn forwards and carried laterally between the cut halves of the thyroid ala. The apparently complete detachment of the cord, together with the ease with which it could be transferred laterally, was specially noticed. Upon, however, the larynx being opened in the middle line, it was found that it was the ventricular band which had been transferred, not the true cord, this showing that accuracy in defining the true cord could not be assured in this manner. Later investigations confirmed the fact that, owing to the varying position (calculated by outside measurement) of the true cords in relation to the centre of the thyroid cartilage, accuracy in location could only be attained by first performing a thyro-fissure.

At the next attempt thyro-fissure was first performed, a triangular piece of cartilage excised (along with the attached cord), when it was found that the anterior end of the true cord was not as free as in the previous case, and could not be transferred laterally on account of the attachment of the perichondrium and muscular fibres of the thyro-arytænoideus. Elevation of the periosteum

in the vicinity released the cord and permitted the piece of cartilage with the attached cord to be easily drawn along the horizontal incision and anchored (fig. 1).

Professor Shattock, to whom I am indebted for co-operating with me and for giving me the opportunity of carrying out these investigations, pointed out that strangulation of the tissues of the cord might result in its being tightly gripped by the cut edges of the cartilage. To avoid this, a small half-circular piece of cartilage was punched out of each half of the cartilage, forming a circular opening in which the cord could lie. In another case, in which the

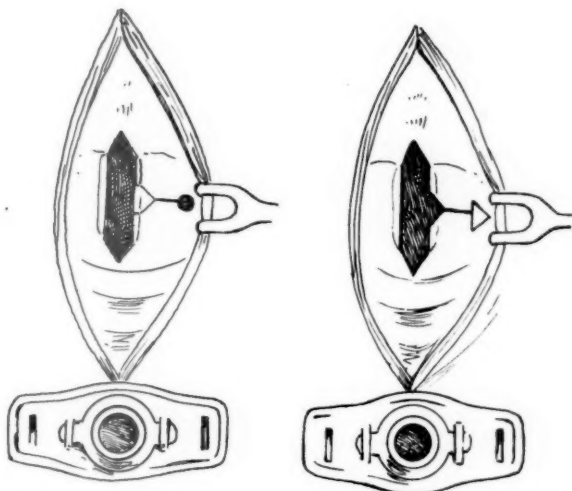


FIG. 1.—Cordopexy. Shows a thyro-fissure followed by antero-lateral transplantation of the vocal cord.

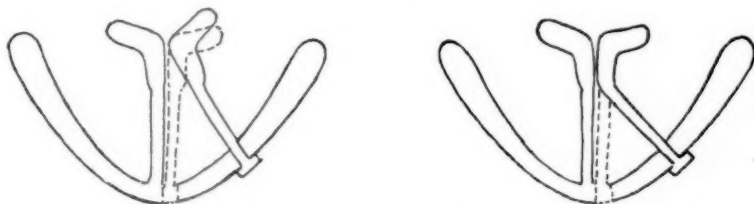


FIG. 2.—Cordopexy. Shows (a) glottic space obtained if the crico-arytenoid joint is motile; (b) the same if the joint is fixed.

ala was ossified, widening the horizontal incision by cutting out a piece with bone forceps removed the constriction of the cord.

By means of this operation the vocal cord or cords may not only be displaced laterally, but also shortened in their antero-posterior diameter, thus increasing the tonicity of the paralysed cord tissues. Transferring a vocal cord one quarter of an inch from the middle line was found to be sufficient to fix it in the position of complete abduction (fig. 2).

## (III) THE VOICE FOLLOWING OPERATIVE PROCEDURES.

Referring to the voice following evisceration or ablation of the soft parts lining the laryngeal box, Chevalier Jackson says that formerly it was believed that permanent loss of voice followed excision of the cord, but in two patients upon whom he operated a fairly loud voice resulted, though very rough and mostly in a monotone. Though a useful voice, it did not compare with the flexibility after thyro-fissure, in which there is unimpaired mobility of the arytenoid joint.

Chevalier Jackson also says that in all the seven successful cases of ventriculo-cordectomy which he performed, the voice was "louder than an ordinary whisper, and loud enough to carry on an ordinary conversation in a reasonably quiet room."

Again, he says the whispered voice, or stage whisper, for which no cord is necessary, will never be lost so long as the respiratory air passes through the larynx, and may be fairly loud, though rough and inflexible.

## (IV) CONCLUSIONS.

If we now have an operative procedure by means of which a permanent cure of stenosis in cases of double abductor paralysis can be guaranteed, the natural airway re-opened, and the patient decannulated, should we not carry this out during the early stage of the paralysis, even at the expense of some impairment of the voice, rather than await the uncertain occurrence of the complete stage of paresis, when the vocal cords may, or may not, assume the recurrent cadaveric position?

Disappearance of the paralysis in bilateral abductor paralysis is an extremely rare occurrence, though it has been reported in a case due to syphilis, and in a few other cases referred to by Chevalier Jackson. Gleitsmann (New York) says there is only one case known of recovery from bilateral recurrent paralysis.

How many cases of double abductor paralysis have been observed, in which all the motor fibres have been destroyed, and the cadaveric stage of the cords reached, and how many cases, having reached this stage, resume the primary stage with the cords in the middle line, which Semon has pointed out may occur later if the adductor muscles recover their tone?

In these cases of double abductor paralysis we have to deal with a very small glottic chink, which is closed more completely on inspiration. The expiration is free, but with strong inspiratory dyspnoea. The voice is normal, or nearly so, but accompanied by a stridulous noise. It is only in the later stage, when the adductor muscles are affected, that the voice is much altered.

## DISCUSSION.

Dr. W. HILL said that in 1908 he had discussed the question of cordectomy for abductor paralysis with Sir Felix Semon, in a case in which he (Dr. Hill) proposed to do arytenoidectomy on the most fixed side. He thought he could do this through the mouth by the direct method. He seized the arytenoid with a large pair of forceps and pulled, but the joint was fixed, and as there was a great deal of bleeding he had to give it up. A few days later, when the œdema which followed had gone down, he performed a laryngo-fissure, and here again an alarming hæmorrhage occurred. He had a tracheotomy tube in the trachea, but the hæmorrhage necessitated plugging of the larynx,

and the operation was never completed. If the operation could be easily performed, he (Dr. Hill) thought it would be the ideal way of dealing with these conditions; but the possibility of an alarming hæmorrhage must be borne in mind. When the arytenoids were removed in any other way, such as for malignant disease, his experience was that the joint was not fixed. But in paralytic conditions there was a post-paralytic contracture, causing what was almost ankylosis, and the operation might be as embarrassing as the one he had related. On theoretical grounds, he thought that the posterior end of one cord was the part to deal with.

Professor S. G. SHATTOCK, F.R.S., said that pathologically or theoretically, the operation of stripping the ventricle or extracting the sacculus would not, in his opinion, produce an effective result, because there would not be enough lateral retraction. If the sacculus were removed, two raw surfaces simply cohered. Even if granulation took place, there would be next to no retraction in the horizontal direction. Members of the Section might be able to help Dr. Irwin Moore with a name for the new operation.

Mr. J. B. CAVENAGH (speaking of cordectomy by the endoscopic method) said this operation could be carried out in two ways, namely, by "tube," or by "suspension." He had recently seen a case in which cordectomy was carried out by the latter method and had been struck by the extreme simplicity and ease with which it was effected with the patient suspended. He thought it must be much more difficult to carry out with the ordinary tube.

Mr. SOMERVILLE HASTINGS suggested to Dr. Irwin Moore that in working out the details of this operation it might be useful, as a first stage, to make a vertical incision in the middle line through the thyro-hyoid membrane. He said he had used such a vertical incision on one recent occasion to remove a growth from the anterior commissure, as it could not be removed by any other method. Through such a button-hole incision the exact level of the cords could be determined, and then a transverse incision could be made through the thyroid cartilage at that level. It was easy to get a view of the larynx from above through such an incision.

Mr. F. HOBDAY referring to Dr. Irwin Moore's statement as to what had been done in the case of the horse, said that he had operated upon nearly 2,500 horses. In the case of hunters this operation had now practically supplanted the old tracheotomy. He was proud that this operation had been suggested as a means of alleviating the condition described as occurring in man. Veterinarians, of course, had much more space in which to do the operation on the horse; they were able to work through the crico-thyroid membrane only, and that was one of the improvements which he (Mr. Hobday) personally claimed to have introduced. As the operation was originally demonstrated to him, the thyroid was cut through with a saw, the incision being large enough to admit four fingers. In the present operation, only a small incision was made, great care being taken to avoid injuring the cartilage; that was one of the reasons the operation had been such a success. In answer to Professor Shattock, he said he had tried to find a dog which had bilateral paralysis, a condition which was found occasionally in the greyhound and other running breeds; but he had not yet succeeded. If he could succeed in performing the operation of ventriculectomy in a small animal like the dog, and bring the specimen to the Section, it would constitute another step towards the performance of the operation in man. Stripping of the ventricle was now done otherwise than with the finger, by means of a small burr with jagged teeth at the edges, which was inserted into the ventricle and slowly twisted in one direction. This everted the sacculus without difficulty. He (Mr. Hobday) liked using the finger better, partly because he was not satisfied with burrs, some of which were made very sharp. Some patterns had to be coated with gauze, so as to enable the ventricle to adhere to them. The cartilage should never be injured, as in the horse that led to ossification. In Italy he had seen the operation performed with the electric cautery. In whichever way the operation was performed it must be done delicately and gently, to ensure success in a large proportion of cases. It had been said that

evening that in the cadaver it was practically impossible to evert the laryngeal sac, and the same was found true of the cadaver of the horse. But when demonstrating it to students, if it was done immediately after the animal was killed, the ventricular lining was easily stripped.

Mr. JAMES BERRY said that twice he had seen bilateral abductor paralysis following thyroid operations. One case was that of a man upon whom bilateral removal of a parenchymatous goitre had been performed by a surgeon in the country. For the ensuing bilateral abductor paralysis another surgeon had done first tracheotomy, then two or three open operations for the removal of the cords in an endeavour to clear the airway. The ultimate result in that case was that another and a permanent tracheotomy eventually became necessary. The other case was that of a young woman with bilateral abductor paralysis which had supervened after he (the speaker) had tied one superior thyroid artery for Graves' disease. Owing to his absence at the war, no further operation had been done for the Graves' disease. Although the physician who had shown him the case had attributed the paralysis to the previous operation on the artery, it seemed very unlikely that this had had anything to do with it. The ligation had been a very simple procedure. Healing had occurred immediately without any trouble, and the laryngeal symptoms did not occur until nearly two years later. The operation that Dr. Irwin Moore had just described seemed a very ingenious one. But, as an old anatomical teacher, he (Mr. Berry) asked whether an objection to the operation did not lie in the fact that the so-called vocal "cord" was not a mere cord, nor even merely the edge of a membrane, but rather the inner and upper border of a triangular mass of tissue containing muscular and other structures in close relation to it. Surely, therefore, it would be difficult to displace the cord outwards against the resistance of this mass of tissue. He would have thought that the mere operation might easily lead to a great deal of chronic inflammatory thickening. He would like to see the operation done, and have a chance of inspecting the patient some time afterwards.

General Sir JOHN MOORE said he would like to amplify the remarks which had been made by Mr. Hobday concerning this operation in horses, particularly with regard to army horses. When he (Sir John Moore) joined the Army thirty-five years ago, the operation for "roaring" consisted of excision of the arytaenoid cartilage, and it was a failure, for in nearly every case there ensued ossification of the larynx. In veterinary practice great care had to be taken in operations on cartilage. Even with tracheotomy he had found great stenosis of trachea afterwards. The present operation for "roaring" in horses, i.e., stripping of the ventricles, was an unqualified success. During the war there were many cases of "roaring," and when a horse had "gone in the wind" it was not of much use for Army purposes; therefore it was cast and sold. That was what happened in the early stages of the war. Later, however, the young officers took up operating, doing the operation which was introduced by Mr. Hobday. Statistics showed that, in one hospital at least, 90 per cent. of the "roaring" horses were returned to active service with very great success.

He (Sir John Moore) said that the following summaries of operations performed at No. 4 Veterinary Hospital, by Major P. M. Edgar, N.Z.A.V.C., would be of interest, as showing the results obtained in the hands of a good surgeon:

#### ROARING OPERATION.

Total number of operations from May 25, 1917, to September 4, 1918	...	128
Number operated on and discharged cured: Left side, 81; bilateral, 30	...	111
Number slaughtered, owing to roaring after operation	...	11
Remaining under treatment, not yet healed	...	6*

111 cured out of 128 represents 86.71 per cent.

\* If any of these were cured the percentage would, of course, be increased.

Case of Pharyngeal Pouch.<sup>1</sup>

By ARCHER RYLAND, F.R.C.S.Ed.

PATIENT, a male, aged 70, complains of gurgling noises in the throat, and of return of particles of food into the mouth, especially on lying down. The history is one of years. There has been no wasting, no disability of any serious kind, and the general health is good. The skiagram is shown, and reveals a large spherical shadow in the position always occupied by a dependent pouch from the upper œsophagus.

## DISCUSSION.

Mr. F. H. DIGGLE (dealing with the phrase that the pouch was in its usual position) said he had two cases under his care, the skiagrams of which he projected on the screen. The first patient was a man aged 58, who suffered from osteitis deformans, with curvature of the spine, and the radiogram was taken obliquely. A small pouch was revealed in the exact mid-line. For the last few months patient had experienced a choking, two or three hours after meals, and coughed up a few bread crumbs. The other patient, a male, aged 68, complained that when he went to bed he could not sleep because of a "gurgling noise" he produced, and again the pouch was exactly in the mid-line. In what percentage of cases did the pouch remain in the middle line? and, if so, on which side of the neck in case of operation should the incision be made? He also asked what operation should be performed. The ostium being in the middle line, it was difficult to think of a two-stage operation. Ought the policy in such cases be to wait in the hope that when the pouch got larger it would be deflected to one side?

Dr. W. H. KELSON said he thought the best course in this case was to leave the condition alone, for evidently the pouch was causing very little inconvenience, and a man aged 70 would probably die of some other complaint. The best guide for making an incision was to pass a probe from above into the pouch and feel its position externally.

Dr. WILLIAM HILL said that these acquired pulsion pouches always originated in the mid-line; and passing through the inferior constrictor it was only the lower end of the pouch which took a direction to one side or the other, and nearly always to the left. All that was required, in order to ascertain its position, was a screen examination from behind. He had had only one case in which the fundus of the pouch went to the right. He offered to operate on that patient, and in that case would have operated on the right side of the neck, but the patient declined. Congenital œroceles passing through the thyro-hyoid membrane, due to a congenital defect, belonged to a different category, and were always laterally situated, passing from one pyriform fossa. Though pharyngeal pouches were rare, nearly two hundred cases of pharyngeal pouch and fistula had been recorded as long ago as 1901. This was stated by Sir Rickman Godlee and the late Mr. Rupert Bucknall in the paper reporting their own case published in that year.<sup>2</sup>

Professor S. G. SHATTOCK, F.R.S., agreed with the suggestion of Dr. William Hill (*see footnote*) that this pouch should be described as pharyngeal. He did not agree that these pouches always arose in the middle line. He knew of one specimen in which the situation of a lateral pouch was immediately behind the posterior border of

<sup>1</sup> This case was described in the agenda as an "Upper Œsophageal Pouch," but at the suggestion of Dr. William Hill the exhibitor agreed that the title should be altered to "Pharyngeal Pouch."

<sup>2</sup> Rickman J. Godlee and T. R. Bucknall, "A Pharyngeal Pouch of Large Size removed by Operation" (with bibliography), *Med. Chir. Trans.*, 1901, lxxxiv, pp. 465-483.



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the thyroid ala, and this exactly corresponded with the fourth branchial pouch, i.e., the condition was of congenital origin, though the size of the pouch showed that its increase was subsequently due to pulsion.

Mr. ARCHER RYLAND (in reply) said that he accepted the suggested term "pharyngeal pouch." By "usual position" he meant the middle line of the neck. No speaker had made any suggestion as to active treatment or operation in this case, and he certainly did not intend to employ any such measures. The patient suffered no real disability, and kept in fact very fit and well.

### **Skiagrams showing Simple Fibrous Strictures of the Œsophagus in a Child.**

By ARCHER RYLAND, F.R.C.S.Ed.

PATIENT, a child, aged 3, in August, 1922, swallowed caustic soda, which was followed by increasing difficulty in swallowing.

November 6: Skiagrams taken and treatment commenced. At this time the child was only able to take liquid nourishment slowly. By œsophagoscopy, the upper stricture was seen and located at 14 or 15 cm. from the upper incisor teeth. The stricture was seen to be small and annular in character, and the lumen was displaced posteriorly. No cicatricial bands or pockets were visible. Dilatation was commenced by means of No. 1 gum elastic urethral catheter.

December 6: No. 12 catheter now passes without difficulty through the upper stricture to a distance of 18 cm. from the upper incisor teeth. The child is now able to take soft solids, e.g., bread and butter, &c.

January 11: No. 12 hollow bougie filled with barium sulphate is seen on the screen to be arrested abruptly at a distance of 18 cm. from the upper incisors. No. 5 bougie enters stomach fairly easily.

### DISCUSSION.

Dr. W. S. SYME said he had had three such cases in the past year, and there were several difficulties in connexion with them. In one of his cases it was impossible to pass a bougie blindly, and when using the œsophagoscope it was easy to see why that was so. The upper portion passed up into the œsophagus, like an *os uteri*; and it was only by passing the œsophagoscope and anchoring the pyramidal portion that a bougie could be passed through the œsophagoscope. He thought such cases would require to be bougied all their life. One of the suggestions made when he showed cases at Glasgow was, that after passing the bougie it would be well to paint the strictured portion with silver nitrate solution. He had done so, and afterwards it had not been necessary to pass the bougie so often as before that application was used.

Mr. ARCHER RYLAND (in reply) said that he had examined this case, in the first place, as far as was possible, with the œsophagoscope, in order to make sure there was no pocket, cicatricial band, or *os uteri*-like condition, complicating the entrance to the upper stricture. In this œsophagus, unfortunately, there were three strictures—one at 14 cm. from the upper incisors, one at 18 cm., and one at 21 cm. The last named was the narrowest and most intractable. He had had some success by making repeated efforts at dilatation with graduated bougies, and proposed to persevere with this treatment at frequent intervals. He did not regard with favour any plan for the retrograde dilatation of the lowest stricture.



**Case of Dentigerous Cyst.**

By M. VLASTO, F.R.C.S.

THIS patient has suffered from a foul tasting discharge and right facial neuralgia since February, 1921. At the operation, on November 22, a sinus was present just behind the right upper second molar. The third molar being unerupted, a probe, passed through the sinus, was arrested at  $\frac{3}{4}$  in. On the evidence of the X-rays, and the absence of nasal discharge, the diagnosis of a follicular odontome was made. The alveolar incision opened up a large cavity, the roof of which was formed by the floor of the orbit, the inner wall by the middle meatal region of the nose and the anterior wall by the very thinned out posterior wall of the antrum. The contained tooth and lining cyst wall were removed and a good deal of the bony margin.

[The patient, the X-ray, tooth and cyst wall, with section of the latter, are shown.]

**Case of Infiltration of Ventricular Band (probably Intra-ventricular Tuberculosis).**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

THE patient, a powerfully built man, aged 42, was first seen in January, 1921, when under sanatorium treatment, complaining of hoarseness of five months' duration; there was then a slight swelling of the right ventricular band, the larynx being otherwise normal. The hoarseness subsided, but returned in September, 1922. There was then found extreme infiltration of the ventricular band on the right side and sub-cordal infiltration on the left. On two occasions the sputum from an induced cough was negative as regards tubercle bacilli; the Wassermann reaction was also negative.

There is at present considerable infiltration of the right ventricular band and some hyperplastic tissue visible in the ventricle. The movement of the right half of the larynx is impaired. The condition is most probably tuberculous.

*Postscript.*—This has since been confirmed by microscopical examination of a minute portion of tissue removed from the edge of the ventricular band.

**Case illustrating the Valvular Action of the Ventricular Bands.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

A FEMALE patient with normal larynx; when she makes a straining effort the edges of the ventricular bands are seen to come into close apposition while the upper surfaces bulge slightly, indicating distension of the ventricles by air compressed in them by the expiratory effort.

**Case of Swelling of the Right Ventricular Band.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

THIS case was previously shown in November, 1922, on account of previous ventricular band phonation. The swelling in this case is most probably of a simple inflammatory nature as shown by the microscopical examination. It had been diagnosed elsewhere as tuberculosis. It is brought forward for comparison with the other case shown at this meeting.

### **Cystic Laryngeal Growth.**

By ANDREW WYLIE, M.D.

H. B., AGED 76, dealer in fish market and formerly accustomed to shout. In 1905 I removed with Whistler's forceps a large growth which was attached to the anterior end of the left vocal cord. Dr. Wingrave described the growth as a "soft fibro-papilloma." Patient has been fairly well since, until the last twelve months. He consulted me to-day, eighteen years after removal, and the condition now found is as follows: A large cystic-looking growth, freely movable, and as far as can be seen, attached to the anterior commissure, but probably it is growing from the same spot on the left vocal cord at which the former growth was found. The growth is anterior to the vocal cords, which are freely movable; the arytenoids are slightly swollen. I intend to remove the growth with a laryngeal snare, by the indirect method.

### **Case of Hoarseness due to Singer's Nodes.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a female. Gradual recovery under rest of voice (restriction to pure whisper) and Curtis's humming exercises. The clinically inflamed tonsils were also removed.

The PRESIDENT emphasized the importance of lessons in voice production, and instanced a case occurring in a music hall singer: the hoarseness had been cured by this means alone without rest of the voice.

## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### Multiple Papillomata of the Larynx.

By H. J. BANKS-DAVIS, M.B.

MALE, aged 37. Thirty years' duration. A tracheotomy was performed by Sir Charters Symonds, for laryngeal papillomata, when the patient was aged 6, but he has had papillomata ever since. The laryngeal symptoms were considerably aggravated during war services. The growths, which almost resemble nasal mucous polypi in their profusion, can be seen to arise from the posterior surface of the epiglottis, the anterior commissure, the vocal cords, and subglottic area as well. What is the prognosis now, and what is the treatment?

#### DISCUSSION.

Dr. D. R. PATERSON said he found that the best treatment for multiple laryngeal papillomata was removal with forceps. He had not heard that the expectations as to the value of radium had been fulfilled in these cases; in one American clinic it seemed to be regarded as a danger than otherwise. If the affection had been present a long time he did not think that much improvement in the voice could be expected, especially in a case in which papillomata were numerous.

Mr. MARK HOVELL said that he had removed many papillomata by the indirect method, in some cases when the larynx was almost completely occluded. With regard to restoration of the voice, he had recently seen a male patient who had had papillomata partially removed by Sir Morell Mackenzie. He (the speaker) had removed all the papillomata, and the removal was followed by complete restoration of the voice. The return to a normal voice gave rise on one occasion to some difficulty in identification of the patient in connexion with claiming deeds, since he had previously been described as having a very hoarse voice.

Sir WILLIAM MILLIGAN said he pleaded for the use of radium in this case, and expressed surprise at Dr. D. R. Paterson's remarks. He regarded papillomata in the larynx as locally infective; it was often difficult to remove them and keep the larynx clear. In some cases radium acted like a charm. Much depended on the way in which it was used; dosage was still somewhat empirical, but more exact than formerly. He exhibited a tube which he used for suspending the radium accurately in the larynx. He was so much impressed with the excellent results obtained, that he scarcely ever used anything else for these cases. He had now in preparation a new instrument designed to prevent the necessity for tracheotomy, and he would exhibit it at a future meeting. It was important in these cases to avoid cutting, tearing, or even cauterizing instruments, because the resultant scarring might interfere with the voice.

Mr. NORMAN PATTERSON inquired as to the dose of radium and the length of exposure which Sir William Milligan employed.

Mr. HERBERT TILLEY asked what kind of screen Sir William Milligan used for the applicator. He also was surprised to hear Dr. D. R. Paterson's remarks about radium: since he (the speaker) understood that it had given great satisfaction in American clinics.

Dr. H. SMURTHWAITE asked whether Sir William Milligan used radium for these cases as a routine treatment, e.g., would he employ it in the case of a solitary papilloma of the cord which could be removed in the ordinary way with fair probability of cure? He (Dr. Smurthwaite) had a patient, aged 26, who since 3 years of age had been voiceless. A year before he had removed a large sessile growth with forceps; since the removal there had been no recurrence, and the voice was now fairly good.

Sir WILLIAM MILLIGAN, answering the questions, said that the dosage of radium must be considered in relation to the nature and extent of the growths. If these were extensive, a large dose, 20 to 30 mg., should be applied for a short time, e.g., two hours. The screen he used was made of silver or of platinum. His remarks had been on multiple papillomata, not single growths. He would not say radium was his routine treatment for the condition, but it was a very valuable adjunct.

Mr. H. J. BANKS-DAVIS (in reply) said that the patient, whom he first saw a year ago, was in the Civil Service, and his (the speaker's) opinion was asked as to whether there was a likelihood of the growth ever becoming malignant. The patient periodically coughed up papillomata, and thus temporarily obtained a freer airway.

### Specimen from Case of Multiple Papillomata of the Nose.

By H. J. BANKS-DAVIS, M.B.

MALE, aged 64. Papillomata of twelve years' duration. Frequent removals. This patient, whose case was reported by the exhibitor in 1912,<sup>1</sup> shows identically the same condition now as he did then. The papillomata bleed readily. The exhibitor suggests radium treatment, or failing this an extensive anterior nasal operation.

*Pathological Report.*—Sections show a highly cellular papilloma of nasal epithelium.

What is the prognosis now, and what is the treatment?

#### DISCUSSION.

Mr. G. W. DAWSON, referring to the severity of these tumours, reminded members that Sir StClair Thomson had stated that only about fourteen cases were recorded in the literature.<sup>2</sup> He (the speaker) had shown a case at a meeting of the Section in 1918,<sup>3</sup> in which the papilloma was attached to the septum. It was of hard consistency and purple in colour. There was no recurrence after removal. He again exhibited the specimen to-day.

Mr. LAWSON WHALE reminded members that he also had exhibited before the Section in 1922<sup>4</sup> a specimen of a large papilloma of the nose. He had searched the literature and found that they were extremely rare; he could only find three cases recorded, two of them in American literature.

Sir WILLIAM MILLIGAN said he would suggest in this case that half a dozen of Stephenson's minute tubes filled with radium should be embedded and left *in situ*.

<sup>1</sup> *Proceedings*, 1912-13, vi (Sect. Laryng.), p. 45.

<sup>2</sup> "Diseases of the Nose and Throat."

<sup>3</sup> *Proc. Roy. Soc. Med.*, 1918, xi (Sect. Laryngol.), p. 132.

<sup>4</sup> *Ibid.*, 1923, xvi (Sect. Laryngol.), p. 12.

**Ventriculo-chordectomy for Double Abductor Paralysis.**

By WALTER HOWARTH, F.R.C.S.

PATIENT, a female, aged 45, married. The patient has been wearing a tracheotomy tube for the past twelve years since an operation on the thyroid gland. The cords were completely adducted, and the ventricular bands always closely approximated. The voice was very weak.

Operation, January 26, under suspension laryngoscopy: The whole of the right vocal cord was removed. It was not necessary to punch it away piecemeal, as when the anterior end was freed the cord tore away cleanly from the underlying muscle, just as if one were tearing the edge off a letter-card. The attachment to the vocal process was cut through and the cord removed whole. The ventricular band was then punched away, particular attention being paid to the under surface.

The tracheotomy tube was removed at the end of a week, and has not been worn since. The voice is a great deal stronger than it was before the operation.

*Further Notes.*—This case is not as yet completed. Since the agenda notes were written there has been some contraction of the airway owing to the formation of a fibrous cord along the cut edge of the muscle. This will be removed. Members will note that removal of the ventricular band exposes the ventricle and enables one to look into it.

**DISCUSSION.**

Dr. IRWIN MOORE said that the operation which Mr. Howarth had performed did not appear to be that which Chevalier Jackson had described as "ventriculo-chordectomy." Jackson's success consisted in punching out not only the ligamentous portion of the vocal cord, but also the muscular tissue constituting the supporting floor of the ventricle. Its non-removal would explain the want of initial success in the present case. Previous authors have shown that, in removing the ligamentous portion of the cord alone, failure resulted from the formation of granulation tissue and cicatrices. Chappel (New York) in 1917 recorded the case of a patient, aged 45, upon whom he operated a few weeks after the onset of double abductor paralysis by removing the ligamentous part of the cord with the aid of suspension laryngoscopy and scalpel and punch. The operation was a failure, insufficient breathing space being obtained, so he cut the opposite recurrent laryngeal nerve, which was followed by the cadaveric position of the cord, and three weeks later he was able to de-cannulate the patient. Chappel's case suggested that, in some cases, it might be advantageous, in the early stage of the disease, to consider the question of cutting the recurrent nerve, prior to more radical operation.

Mr. E. MUSGRAVE WOODMAN congratulated Mr. Howarth on what he had done. He himself had three weeks ago performed the external operation for chordopexy as suggested by Dr. Irwin Moore at the last meeting of the Section—on a case of double abductor paralysis, but at the moment it was not a great success. The operation was followed by a prolonged and severe coughing. When viewed a week after the operation the cord was seen as in its new abducted position but the glottic space anteriorly was largely blocked by granulations. He considered it important to avoid cutting away the small piece of cartilage, in which the anterior end of the cord was inserted, so as not to leave an opening in the thyroid cartilage through which granulations could protrude into the larynx.

Mr. E. D. DAVIS said that he must congratulate Mr. Howarth on the success of his operation, but he would like to see the patient again in three months, because in so

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many of these cases scar tissue formed and re-insertion of the tracheotomy tube was necessary. He did not think there was sufficient room in the patient's larynx for her to breathe comfortably, and she was not now embarrassed because her tracheotomy fistula was still patent and was being used for respiration. One of Crile's colleagues treated these cases by raising a flap of mucous membrane above the cord, and another flap below, and after excising the vocal cord, he stitched the mucous membrane together. Very little scarring followed and the results were said to be good.

Mr. WALTER HOWARTH (in reply) said that he showed the case in a transition stage. The operation was performed only five weeks ago. It was not easy to get an idea of an operation unless it was seen performed. When he was in America, Chevalier Jackson was not doing these things. He had written to Dr. Jackson with regard to the operation and had followed the description as carefully as possible. He had punched out the floor of the ventricle in the present case, and he thought that a sufficient scarring of the ventricular band had resulted. He did not remove enough of the vocal cord musculature and there was now a fibrous cord impinging on the airway. Next week he intended to punch away the rest of the cord up to the thyroid cartilage; which would give sufficient respiratory airway. With regard to cutting the laryngeal nerve in early cases, the present case was not an early one; the patient had been wearing a tracheotomy tube for ten years; so that the procedure would scarcely be applicable. He would show the case again later.

### **Laryngostomy for Complete Subglottic Stenosis.**

By WALTER HOWARTH, F.R.C.S.

THE patient, a male, aged 12, had worn a tracheotomy tube ever since a high tracheotomy for diphtheria in early life. The cricoid cartilage had been divided, and had fallen in.

Operation, 1913: Laryngo-tracheostomy. The gutter into which the lower part of the larynx and the upper part of the trachea was converted was kept open for many months by the use of a large rubber tube, on to the back of which a sheet of oiled silk was sewn. The cavity was packed daily on to this and eventually the epithelium growing in from the surface covered the whole area. The epithelialized groove was then covered in by a plastic operation. Unfortunately the war intervened before the final operation, and the patient was lost sight of until a few months ago.

#### DISCUSSION.

Sir WILLIAM MILLIGAN said that great skill and patience had been exercised over this case; the daily dressing for so long a time was very irksome. Had the exhibitor ever tried direct grafting in these cases, so as to avoid this constant dressing; if so, had it succeeded? He himself had tried grafting once, but it was a failure.

Mr. E. D. D. DAVIS said he had once tried grafting in a similar case, but the graft soon came away. It would not adhere to the interior of the larynx.

Mr. G. W. DAWSON said that one of his colleagues also had tried a graft, but it had not been satisfactory.

Dr. BEEVOR said he was reminded of a case in which the trachea of a large dog had been used as a graft, and the operation succeeded very well. The death of the patient some time afterwards had been due to another cause.

Mr. HOWARTH (in reply) said that he had not carried out grafting in this case, because when the operation was performed ten years ago grafting in the air passages was not so freely done as at the present time. If in the future he should have a similar case he thought he would try direct grafting. He understood from Mr. Harmer that there was a good chance of grafts succeeding in certain cases of this type.

### **Hæmorrhagic Angiosarcoma of Upper Jaw.**

By H. J. BANKS-DAVIS, M.B.

MALE, aged 60. Operation. Recurrence. Apparently cured after three exposures to X-rays by Erlangen method (Dr. Morton).

This patient was admitted into the West London for severe hæmorrhage following the "snaring of a nasal polypus" in another hospital out-patient department, two days previously.

The cheek rapidly swelled, and an exploration of the antrum showed that it was occupied by a fleshy vascular growth, which expanded the bone even after a free curettage a few days before.

After the first exposure (Erlangen apparatus, eight hours), the swelling, hæmorrhage and other symptoms disappeared. As a precaution, Dr. Morton has thought it advisable to give him two more applications, and the patient's condition is as you see now, apparently cured.

Mr. C. A. PARKER (President) said that the result was very satisfactory; there being at the present time no evidence of malignant disease.

### **Complete Laryngectomy for Malignant Disease.**

By WALTER HOWARTH, F.R.C.S.

PATIENT, a male, aged 54. Operation a year ago. The patient is shown in the endeavour to combat the general belief that the operation is a very mutilating one and that the patient is always miserable. The pharyngeal funnel is well shown, but the pharyngeal voice is much poorer than usual.

### **A Case of Sarcoma of the Nose cured by Radium.**

By E. MUSGRAVE WOODMAN, M.S.

S., MALE, aged 30, grinder. First came under my care early in the year 1921. He then had a large swelling of the nose which had expanded the bone and deviated the axes of the eyes outwards. He was suffering from severe headaches.

The interior of his nose appeared to be the seat of an extensive growth coming down from the base of the skull, which had destroyed the septum and pushed the bones of the nose outwards. A section taken showed it to be a myeloid sarcoma, which was inoperable. At that time no radium was available. A Wassermann test was made and found to be negative.

I kept him under observation and gave him a course of colloidal selenium; but the condition gradually grew worse, the nose broadened and the skin became reddened, adherent and infiltrated.

In December, 1921, I gave him an anæsthetic and introduced a barrage of four 10-mg. tubes of radium between the growth and the skin in different directions and a 50-mg. tube in the substance of the growth. The condition gradually improved and he has had further doses of radium on three different occasions.

The nose has shrunk, the cavity is free from growth but tends to form crusts, and the man is well and at work.

(A microscopical section is also shown.)



## DISCUSSION.

DR. LOGAN TURNER asked how long the radium was kept in position in this case. He (Dr. Logan Turner) had had two or three cases of sarcoma of the nose and nasopharynx treated by radium, apparently with great success. The application was made in each case continuously for five days. The dose varied from 1,700 to 2,000 milligram-hours, and the pure salt was used, not emanation tubes. Dr. Dawson Turner, of the Radium Department, Edinburgh, was a supporter of long-continued irradiation.

MR. NORMAN PATTERSON said that he deprecated the use of the word "cure" in these cases when only a year or two had elapsed. He thought there were still some suspicious lumps about the base of the nose.

MR. J. F. O'MALLEY asked whether Mr. Woodman had ever known destruction of bone to follow the use of radium. He had recently had a case which had been under treatment for two years, and the patient had seemed to be approaching a cure, and therefore was allowed to return home. A few weeks later he died of brain trouble, though there was no sign of recurrence in the nose.

MR. HERBERT TILLEY said that he also objected to the use of the word "cure" in these cases. He had seen a patient 16 months before with a large sarcoma of the left ethmoidal region, proptosis of the eye on the same side, intense neuralgia, a blood-stained discharge, complete nasal obstruction, and tinnitus in the left ear, &c. Patient had been treated by the intensive X-ray method, and the condition disappeared in six weeks, so that he could breathe freely through both nostrils. Three months ago he (the patient) was suffering from indigestion, was getting very thin, and was found to have a growth in the stomach. It seemed to be the almost universal experience that while irradiation destroyed the primary malignant growth, distant metastases occurred later.

MR. MUSGRAVE WOODMAN (in reply), after exhibiting slides of the case by means of the epidiascope, said that he considered that the word "cure" had rightly been criticized. It was forgotten, however, that this was not a case of round-celled sarcoma, but a giant-celled growth, and a myeloid sarcoma was the "plaything" of malignant disease. The lumps to which Mr. Norman Patterson had referred had proved under the microscope to be granulation tissue. They were associated with some necrosed bone, which had followed the use of radium, and would separate so slowly that there would be a sufficient protection internally to prevent the danger of meningitis. The longest time he had left radium in position was twenty-four hours; each time 10 mg. were buried between the bone, and a 50-mg. tube inserted into the substance of the growth.

**Tuberculous Ulcer of the Dorsum of the Tongue.**

By WALTER HOWARTH, F.R.C.S.

MALE, aged 24. The patient suffers from pulmonary tuberculosis. Deep ragged ulcer in tongue of three months' duration, not causing any discomfort and healing under treatment. Masses of giant cells seen in the section. Wassermann reaction negative.

**Extensive Lupus of Palate, Pharynx and Larynx.**

By WALTER HOWARTH, F.R.C.S.

FEMALE, aged 15. The patient has had several operations for cervical adenitis in 1918 and 1920. Loss of voice four months. No pain or discomfort. There are some signs of pulmonary tuberculosis.

**Laryngeal Case for Diagnosis.**

By H. BUCKLAND JONES, M.B.

MALE, aged 42. Complaints of hoarseness, which started about January, 1922. Attended a chest hospital recently for about two years. Sputum examined negative. Losing weight. Reports of blood and sputum tests which are being taken will be submitted at the meeting.

**Tuberculoma of the Pharynx.**

By NORMAN PATTERSON, F.R.C.S. and G. C. CATHCART, M.D.

FEMALE, aged 46. Complaints of a lump in the back of the throat and a swelling in the neck. Duration of trouble uncertain. The patient's posterior pharyngeal wall is occupied by a very extensive irregular swelling presenting a granular appearance. A portion removed showed the condition to be a tuberculoma. Examination of the chest is negative. Radiographic report: "A few calcareous nodules at the hilum of the right lung. No evidence of pulmonary disease."

**DISCUSSION.**

Mr. H. J. BANKS-DAVIS remarked that Mr. Howarth, in his first case, referred to "healing under treatment." He (the speaker) considered that the best treatment for tubercular ulcers on the tongue was the application of pure chromic acid. If there was pain, the chromic acid formed an albuminate over the tongue and quickly relieved the pain. And again the chromic acid, if applied in full strength, healed up the ulcer rapidly. Anything less than a 40 per cent. solution he thought would not have the effect he referred to.

Sir STCLAIR THOMSON, referring first to the case of tuberculous ulcer of the tongue exhibited by Mr. Howarth, said that an annotation had appeared in the *Lancet* last year stating that this was one of the most rare complications of pulmonary tuberculosis, and that the ulcerative form only occurred in advanced cases and was nearly always fatal. He exhibited the drawing of a similar case in a male patient who was admitted to a sanatorium with tubercle bacilli in his sputum; it was a "three-lober case." He was also suffering from tuberculosis of the larynx. The Wassermann reaction was negative. The drawing showed the favourite site, viz., on the tip and sides of the tongue. As was remarked in the *Lancet*, these lesions were more common in men than in women. He gave this man, in 1917, a solution of chromic acid, 20 gr. to the oz., to use as a paint. After his regulation three months in the sanatorium, his condition was *in statu quo*. Examination of the patient last year showed that the tongue and larynx were healed. Patient walked, cycled and dug now, and he had been accepted for insurance as a first-class "life." Mr. Howarth's second case was an extensive case of lupus, and he thought the quickest cure was to do a tracheotomy; the resultant rest would greatly benefit both larynx and pharynx. He also recommended the galvano-cautery and sanatorium treatment. Referring to the case shown by Mr. Buckland Jones, he (Sir StClair Thomson) thought it was a very promising one. He had seen cases of lupoid tuberculosis limited to the pharynx, in which no disease in the lungs could be detected. Frequently there were glands, which were not usual in laryngeal cases. Diathermy left an extensive scar, and half a dozen applications of the galvano-cautery would be better at monthly intervals.

## 52 Patterson and Cathcart: *Tuberculoma of the Pharynx*

Dr. LOGAN TURNER said he thought that the base of the tongue was infected in Mr. Howarth's case of extensive lupus of the palate, pharynx and larynx. It was very rare to see lupus of the tongue. Though he had seen many cases of lupus he had only once seen the tongue involved. The epiglottis was diseased in a large number of cases.

Sir WILLIAM MILLIGAN said he supported the plea for rest in this type of case and advised not only that the larynx should be given physiological rest by the performance of tracheotomy, but also that the patient should undergo the "silence cure," just as if it were laryngeal tuberculosis. He had found "silence" of value in carcinoma of the larynx, in improving the condition for a time, and it was of value in almost every laryngeal condition. In some of these lupus cases he had had good results from tracheotomy and in others from the application of the galvano-cautery. He believed also that Mr. Buckland Jones's case was tuberculous.

Mr. HERBERT TILLEY said he would suggest that if the proposed measures failed, tuberculin should be tried, and related the case of a young girl who had had various measures tried for this condition, including punching out portions of the anterior pillars of the fauces. Recurrence took place on the upper surface of the soft palate. It then spread down to the epiglottis and larynx. In despair of surgical treatment he suggested tuberculin, and it was continued for three months. At first the reaction was severe but gradually became a diminishing feature, and after six months it was impossible to see any sign of disease in the whole area which had been implicated. He had never seen a case of the kind so dramatically cured.

Dr. H. SMURTHWAITE said he had had a case which had been much benefited by rest to the voice, following tracheotomy for acute stenosis. The larynx had been blocked with tubercle and there had also been a secondary infection, which might have been responsible for the acute stenosis. In three months the condition had cleared up, except in the interarytænoid region. There had also been marked tuberculosis of the left lung, and the patient was sent to a sanatorium, but he (Dr. Smurthwaite) did not know the later results.

Mr. W. H. JEWELL said that seven years ago he had seen a case of lupus of the larynx in which he had to perform tracheotomy. The cords were so glued together that they constituted a complete diaphragm, which still existed, except for a small perforation, and the tracheotomy cannula had to be continually worn. Five years ago under a suspension laryngoscopy he attempted to separate the cords, but found them so firmly adherent that only a small opening could be made, and this had since remained patent. He thought that the opening could be still further enlarged now that the lupus was cured.

Mr. NORMAN PATTERSON (in reply) said that he considered the swelling on the left side of the neck in his case was not an enlarged gland, but a sebaceous cyst; because it was attached to the skin, was freely movable, and had a long history. He would remove the swelling, and have it examined microscopically.

Mr. HOWARTH (in reply) said that in the case of tuberculous ulcer on the dorsum of the tongue, he had merely applied 2 per cent. iodine in spirit. He had been to one sanatorium, and was now in Victoria Park Hospital, waiting for admission to another. The lupus case was now in hospital for pulmonary tuberculosis. He would suggest tracheotomy to the patient, for the purpose of securing rest to the part. It should not, however, be forgotten that these cases showed a great tendency to get well of themselves. Ten years ago he showed a similar case which cleared up entirely on the patient being sent to Margate and being given good food for a period of several months.

P.S.—The condition has since been treated by diathermy. The "tumour" in the neck was removed, and proved, on microscopic examination, to be a sebaceous cyst.

## Improved Antrum-exploring Trocar and Cannula.

By H. M. WHARRY, F.R.C.S.

THIS is a modification of the ordinary straight trocar and cannula, having two improvements.

(1) The point is guarded by having a small ring placed round the cannula half-an-inch from the end. The ring is very small and does not interfere with manipulation, but when it meets the medial wall effectually prevents the point of the trocar from hitting or transfixing any of the further walls of the antrum at the moment of puncture.

(2) There is an improved joint between the cannula and the nozzle. By means of this the two can be held firmly together by the finger and thumb, making it impossible for them to fly apart when pressure is exerted by the syringe. The fingers can also be held out of the way of any discharge coming from the nose.

### DISCUSSION.

Mr. HERBERT TILLEY warned against the blowing of air into the sinuses before irrigation of a sterile fluid, because one day a patient might die in the consulting room from air embolism, owing to air being injected into one of the blood-vessels of the mucous membrane. In one case in which this had occurred the patient fell from the chair, and had a right-sided motor paralysis with aphasia lasting for twenty minutes. Fortunately that patient recovered. Nine deaths had been recorded as due to air embolism.

Mr. W. H. JEWELL said he had heard of a case of death in a consulting room following the practice just referred to.

Sir STCLAIR THOMSON said he had been taught in Vienna first to blow air through the cannula, and he had never seen an accident from the procedure. Was it certain that none of the cases mentioned were due to cocaine intoxication? It would be important to settle whether air really was responsible.

Mr. CAVENAGH asked how much air was required to cause embolism. Even if one began irrigation with fluid immediately, some air was present in the cannula, and would not that be sufficient to cause an embolus? With regard to the improved antrum trocar and cannula, he remarked that no one would attempt to puncture dense bone without careful calculation of the distance, and by means of the finger grasping and steadying the shaft of the trocar so as to avoid any accident. He considered it doubtful whether the small shoulder on the new cannula was sufficient to dispense with this precaution.

Mr. C. A. PARKER (President) said he had known two instances of accident occurring in cases of atrophic rhinitis, with a very small antrum. In both the cheek had been wounded, and in one an abscess had resulted followed by some necrosis of the lower plate of the orbit. It was an accident that was not altogether rare.

## Case of Ulceration of Palate and Fauces.

By T. JEFFERSON FAULDER, F.R.C.S.

THIS case was presented at the December meeting by Dr. Kelson and Dr. Thornhill, and recently reported in the *Proceedings*<sup>1</sup> to which reference can

<sup>1</sup> Kelson, W. H., and Thornhill, W. H.: "Case of Ulceration of the Palate and Fauces," *Proc. Roy. Soc. Med.*, 1923, xvi (Sect. Laryngol.), p. 13.

be made. The Wassermann reaction was negative. The ulcers of the palate and fauces, three in number, have healed rapidly under simple treatment with thyroid extract and potassium iodide. The patient is putting on weight. The dysphagia, which was a symptom, has been proved by observation in the wards to be partly a neurosis and is in any case relieved.

**Specimen from the Post-mortem Room of a Large Cyst of the Orifice of the Larynx arising from the Arytæno-epiglottidean Fold.**

By E. D. D. DAVIS, F.R.C.S.

A WOMAN, aged 50, was sent to the hospital for hoarseness of three years' duration. Dyspnoea and stridor commenced a few weeks before observation. A large pale smooth cyst filled the orifice of the larynx and obscured the view of both vocal cords. It was sessile, and was growing from the inner surface of the right arytæno-epiglottidean fold and epiglottis. On August 1 the cyst was punctured by indirect laryngoscopy, but it was difficult to punch out a piece of the cyst wall. On August 8 the cyst had filled again and she was given an anæsthetic (C.E.) and an attempt was made to remove the cyst by suspension laryngoscopy, but the view obtained was unsatisfactory and the cyst was only punctured.

December 27, 1922: The cyst had filled again and the patient was more stridulous. It was decided again to attempt removal by suspension laryngoscopy and if that failed to do an external operation. She took the anæsthetic badly, with signs of laryngeal obstruction, and in getting her into position the stretching of the neck stopped the breathing and a hasty laryngotomy was done, but the patient collapsed and died.

The larynx shows a large biloculated cyst which was seen at the posterior border of the thyro-hyoid muscle as soon as the neck was opened. This portion of the cyst freely communicated through the thyro-hyoid membrane with the cyst within the larynx. The stretching of the neck expressed fluid from the outer portion of the cyst to that within the larynx, and the post-mortem examination disclosed obvious signs of asphyxia.

DISCUSSION.

Dr. IRWIN MOORE did not think this was an ordinary position for a cyst. It appeared possible that it had originated from the sacculus laryngis and passed through the roof of the ventricle, and later extended through the thyro-hyoid membrane.

Mr. HERBERT TILLEY demonstrated by means of the epidiascope a large laryngeal cyst, which was found in a man, aged 40, who had been nearly suffocated by it. This was prevented by the patient passing in his finger and rupturing the cyst. Twice the speaker cut away most of the cyst wall and cauterized it, but it recurred, together with swelling under the chin, which later suppurated. Mr. Trotter evacuated the pus and mucus, and then dissected out a large cyst, which communicated through the thyro-hyoid membrane with the cyst in the larynx.

Mr. E. D. D. DAVIS (in reply) said that the swelling in his case contained fluid, and he therefore thought that it was a mucous retention cyst, commencing on the anterior surface of the epiglottis. There was no laryngocele.

**Case of Outgrowth from the Ventricle in a Subject of Pulmonary Tuberculosis.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

A FEMALE patient, aged 19, first seen by me in January, 1922, on account of hoarseness of six months' duration. A smooth reddish rounded outgrowth projected from the anterior part of the left ventricle, lying on the surface of the vocal cord; the edges of the vocal cords were at that time irregular. She was ordered abstinence from the use of the voice and the habitual wearing of a creasoted respirator. She has improved steadily and there does not seem any call for more active treatment at the present time, though the outgrowth is still present.

**DISCUSSION.**

Dr. IRWIN MOORE said he thought this outgrowth was a tubercular hyperplasia of a fold of mucosa lining the sacculus laryngis, as proved to be the case in a specimen in the Museum of Golden Square Hospital, which he had investigated along with Professor S. G. Shattock in connexion with his (Dr. Irwin Moore's) paper on "So-called Prolapse of the Laryngeal Ventricle."<sup>1</sup>

Mr. CAVENAGH asked whether this was not a case of soft fibroma. If the patient had not had tuberculosis what would the present growth in the larynx have been considered to be?

**Mounted Specimen showing Two Foreign Bodies—one movable and the other fixed, in the Trachea of a Child, aged 3.**

By H. J. BANKS-DAVIS, M.B.

THE fixed body was the cause of the symptoms, the movable body was the cause of death. This beautiful specimen, probably unique, was mounted by Dr. Elworthy. It shows two fine, translucent flakes of bone from the scapula of a rabbit which were inhaled during the act of swallowing. The scales of bone are shown in the specimen in the positions in which they were found—one above the tracheotomy opening and the other below it. Sudden death occurred after a fit of coughing. Bronchoscopy revealed nothing except paralysis of the left vocal cord, under which the upper scale of bone was impacted. The clinical history of the case is one of great practical importance.

**Mounted Specimen showing a Threepenny-piece impacted in a Perforation between the Œsophagus and Trachea of a Baby, aged 3 months.**

By H. J. BANKS-DAVIS, M.B.

COIN removed by tracheotomy, but death resulted.

The report accompanying an X-ray plate taken by Dr. Morton stated: "The coin was in the trachea as it moved up and down on respiration."

<sup>1</sup> *Journ. Laryng. and Otol.*, 1922, xxxvii, pp. 265, 333, 381.

From a comparison of the size of a threepenny-piece with that of the glottic aperture of a child aged three months, it seemed to me impossible that the coin could have dropped through the airway as stated. On passing a tube into the œsophagus I thought I saw the edge of the coin, but it disappeared on my attempting to grasp it; on passing a tube into the trachea I thought I detected the edge of the coin but failed to secure it. The fact is that the coin was slipping through from the œsophagus into the trachea and from the trachea into the œsophagus and so eluded capture whenever an attempt was made from either locality to secure it. Eventually I removed the coin by a low tracheotomy when I observed the edge of the coin presenting in the œsophageal wound.

The child died three days later, probably from œsophageal secretions draining into the trachea and causing broncho-pneumonia.

My colleague, Dr. Irwin Moore, kindly assisted with his instruments in this case.

#### DISCUSSION.

Dr. IRWIN MOORE referred to the rarity of this case, and to the value of the specimen for teaching purposes. In a paper which he had contributed to the *Lancet*, in 1919,<sup>1</sup> he recorded the fact, from a search of the literature, that only thirty-seven coins had found their way into the air-passages, during a period of nearly a century (from 1819 to 1915). Of these, twelve were impacted in the bronchi, and three in the trachea, the remainder being in the larynx, and he was unable to find a case similar to the one now reported in which a coin had penetrated from the œsophagus into the trachea. One of the coins aspirated into the trachea was recorded by Chevalier Jackson, in 1915, in the case of a child, aged 3, who had a nickel coin measuring 21 mm. impacted in the trachea for one day. It was surprising how a coin with this diameter could enter the trachea of a child of this age. In the case now recorded by Mr. Banks-Davis the insertion of a tracheotomy tube caused complete obstruction to respiration owing to displacement of the coin from the tracheo-œsophageal wall into the lumen of the trachea, and but for the immediate insertion of a bronchoscopic tube into one of the main bronchi death would have occurred at the time. This case demonstrated the importance of having a bronchoscopic tube always ready as an alternative or adjunct to tracheotomy, in all cases of respiratory obstruction.

Mr. BANKS-DAVIS (in reply) said the radiographer had reported that the coin "was in the trachea." He (the speaker) thought it impossible that a threepenny-piece could pass between the cord in so young a child; it did not occur to him that it had ulcerated through from the œsophagus and really occupied a portion of both passages.

### Tumour of Nasopharynx.

By F. CUNLIFFE ORMEROD.

A. C., MALE, aged 25, has felt a "clogging" of the throat for the last two years. Nothing of note in nose or oropharynx except slight forward displacement of soft palate. Posterior rhinoscopy displays a sessile tumour which appears to be attached to the posterior part of the right lateral wall of the nasopharynx. The choanæ are free and there is no nasal obstruction. The tumour is firm in consistency, but not particularly hard. The only history is that of diphtheria and tonsillitis. No history of epistaxis was obtained.

Mr. A. J. M. WRIGHT thought this was a cyst or a lipoma.

<sup>1</sup> *Lancet*, 1919, ii, pp. 566, 609.



**Tumour of the Larynx; ? Malignant.**

By W. H. JEWELL, O.B.E., M.D.

W. D., MALE, aged 37. Hoarseness, four years. The infiltration obscures the vocal cords; on the left side it is succulent and extends the whole length of the glottis involving the ventricular bands, aryepiglottic fold, and pyriform fossa. On the right side it is less extensive and appears drier. Left side is fixed, slight movement right side. Obstruction of glottis, except a narrow posterior chink, necessitating a tracheotomy. Wassermann negative; sputum negative for tubercle bacillus. Glands infected in the left submaxillary region.

**Swelling on Posterior Wall of Pharynx.**

By FREDERICK SPICER, M.D.

MALE, aged 13, with swelling of the left half of the posterior pharyngeal wall, which followed enucleation of the tonsils two years ago. Opinions are invited as to diagnosis.

DISCUSSION.

Mr. H. J. BANKS-DAVIS said he thought the swelling was an enlarged retro-pharyngeal tubercular gland.

Mr. NORMAN PATTERSON and Dr. IRWIN MOORE agreed with Mr. Banks-Davis.

**Clonic Spasm of the Palate.**

By DAN MCKENZIE, M.D.

PATIENT, a female, aged 29. After a prolonged attack of acute rheumatism she became aware of a "clicking" sound which seemed to be produced in the throat. Examination shows a regular clonic contraction of the soft palate, posterior and lateral pharyngeal walls, and base of the tongue, i.e., of the muscles concerned in swallowing. The movement occurs about 150 times in the minute, and is most pronounced when the mouth is opened and the pharyngeal apparatus is, as it were, on the balance between normal rest and the act of retching. It is to be noted, however, that the movements occur also when the mouth is closed, and they have frequently awakened the patient when asleep. A possible relationship with the Eustachian clicking which seems to be the source of some types of audible tinnitus aurium occurs to one. The larynx is unaffected.

Dr. C. O. Hawthorne, who has kindly examined the case for me, regards the symptom as a functional neurosis.

Mr. W. H. JEWELL said he had shown at a meeting of the Otological Section the case of a child in whom the "click" could be heard two yards away, due to spasm of the soft palate. After removal of adenoids the "click" disappeared.

## Trachea Obstruction due to (?) Arrest of Development of the Trachea.

By C. A. SCOTT RIDOUT, M.S.

THIS case was reported at the meeting of the Section on December 2, 1921.<sup>1</sup> The patient died in May, 1922. The parts removed post mortem were shown at the meeting of the Section on November 3, 1922.<sup>2</sup>

### FURTHER INVESTIGATIONS AS TO THE CAUSE OF THE OBSTRUCTION, AND REPORT BY PROFESSOR S. G. SHATTOCK, F.R.S.

The larynx and trachea, &c., of a boy aged 16. The thyroid gland is uniformly enlarged, the right lobe measuring 10 cm. (4 in.) in the vertical diameter; superiorly it extends to the level of the hyoid bone. The left lobe reaches slightly lower than the right, and to within half an inch of the bifurcation of the trachea. The trachea in consequence of the pressure of the goitre is flattened from side to side for a distance of about 1½ in.

Immediately below the goitre there is a voluminous, lobulated mass of enlarged lymphatic glands: some of these are discrete; others coherent. Inferiorly the mass completely fills the cleft between the bronchi. On the anterior aspect, occupying a median interval in the gland mass, there is a small portion of the normal thymus—a loose, delicately lobulated, soft structure of brownish pink colour. On the right side the upper portion of the thymus is enlarged by diffuse growth, which has almost destroyed its natural lobulation, and seeing that this consists of lymphatic tissue it may be regarded as a lymphosarcoma. The most enlarged portion of the thymus (about the size of a tangerine orange) lies immediately below the right lobe of the thyroid, and against the right wall of the right bronchus, which it aids in distorting. Portions of the thymus on the left side are enlarged to a lesser degree, in the same manner, and lie in front of the left bronchus in the compression of which they likewise take part.

## Demonstration of Specimens of Tumours in the Inter-arytænoid Space of the Larynx.

By W. JOBSON HORNE, M.D.

AT the request of the author the publication of the abstract of his demonstration, together with the report of the discussion upon it, has been deferred to a later issue.

THE following case has been referred for later publication until further investigations, or a completed report, have been submitted:—

G. W. DAWSON, F.R.C.S.I.: "Infiltration and Ulceration of the Epiglottis and Right Ary-epiglottic Fold."

<sup>1</sup> *Proceedings*, 1922, xv (Sect. Laryngol.), p. 13.

<sup>2</sup> See *Proceedings*, 1923, xvi (Sect. Laryngol.), p. 10, for a description of the specimen. The specimen (not yet numbered) is now included in the Museum of the Royal College of Surgeons.

## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### Two Cases of Laryngo-fissure for Intrinsic Cancer of the Larynx.

By Sir STCLAIR THOMSON, M.D.

*Case I.*—A judge, aged 80, shown six months after operation. The right vocal cord was replaced, except for a small portion posteriorly, by an irregular, pink, fleshy growth. The cord moved freely but the clinical appearances were typical of malignant disease, and the condition had already been diagnosed as such by Dr. Permewan, of Liverpool. Patient has smoked all his life and still smokes three or four ounces of tobacco every week.

Laryngo-fissure October 21, 1922; thyroid ala removed; tracheotomy tube left *in situ* seven hours. Patient swallowed easily the same evening and was sitting out of bed next day, playing chess. He returned to Wales eighteen days after the operation.

The chief interest of this case consists in the age of the patient and in the absence of shock. It was only after careful consultation that it was decided to operate, Dr. Frederick Price reporting that there was fibroid degeneration of the myocardium, arterial disease, probably a rather dilated aorta, and attacks of angina during the last five years.

Since the operation the patient has been out hunting, sometimes following the hounds for three or four hours.

*Case II.*—A male, aged 40, shown one year after operation.

This case is interesting in view of the comparatively early age of the patient and because of some difficulty in diagnosis.

In January, 1922, the patient lost his voice suddenly when trying to sing. In March when first seen by exhibitor the right vocal cord was found to be invaded in its whole extent by an irregular, mammelated, projecting cauliflower growth with white points. The cord moved freely; examination for tubercle gave negative results. Diagnosis of epithelioma was made from clinical appearances, and was agreed to by Messrs. Tilley and Hope. Voice rest for six weeks resulted in improvement of voice but in a more marked local condition.

Laryngo-fissure, March 19, 1922, with removal of thyroid ala. Growth removed *en masse*. Profuse hæmorrhage occurred at the end of the operation and some spouting vessels were ligatured. The operation occupied one and a quarter hours, but forty-five minutes more were spent in checking the bleeding. There was no subsequent hæmorrhage and the tracheotomy tube was removed the same night.

On Patient sat up next day; went out for a walk on the fifth day; and on the twelfth day he assisted at a laryngo-fissure on another patient and left for the country the same afternoon.

The voice is, at present, not as satisfactory as in most cases, probably due to his failing to report himself from July to September, and to over-use of

his voice. This started a compensatory hypertrophy in the ventricular band before a new cicatricial cord had formed. He was, therefore, put back to whispers for two months and the voice is now improving under lessons from Mr. MacMahon.

**Laryngeal Case apparently of Epithelioma (possibly Syphilis).  
Completely healed and arrested under X-ray Treatment  
without Operation.**

By Sir STCLAIR THOMSON, M.D.

MALE, A. C., aged 68, first seen October, 1921, complaining of gradual loss of voice for nine months. The right vocal cord was quite fixed; the whole being deeply ulcerated and mouse-nibbled, and with a succulent and indolent appearance very suggestive of tubercle. Though suffering from bronchitis no symptoms were present in his lungs to justify a diagnosis of tubercle. Wassermann reaction negative. Blood-pressure 150, auricular fibrillation, no temperature. Weight, 9 st. 10 lb. He had been getting thinner.

The local condition became more marked in November, and a month later was more suggestive of malignant disease. The right side of the larynx, except the arytaenoid, was still quite fixed; the cord was invaded throughout its entire length; there was a small red granulation in the anterior commissure and a suspicion in the right inter-arytaenoid region. The ventricular band and the arytaenoid were not invaded.

The case was seen by Mr. Trotter, who said that, in view of the extension and the patient's age and feebleness, operation was not justifiable. In January, 1922, patient reported that he had seen Sir James Dundas-Grant who had removed a portion of growth endolaryngeally which was reported to be malignant. On January 17 the condition seemed worse. There was a gland the size of an almond on the right side of the neck; the right side of the larynx remained quite fixed, the arytaenoid was enlarged and infiltrated with a slight defect at its summit. The right ventricular band was red and infiltrated, and concealed a good deal of the right cord, and there appeared to be subglottic extension.

The patient was transferred to Dr. Knox for X-ray treatment.

By June, 1922, patient was looking better; had gained weight; no gland in neck. The appearances of the larynx had changed in a most remarkable way. The right arytaenoid was quite normal and as good as the left; it moved, but the right side of larynx remained fixed by infiltration of the ventricular band. Still, this was diminished so that the anterior third of the right vocal cord was visible, and was seen to be quite healthy. The posterior two-thirds of the cord, on its upper and inner surface, was occupied by a clean, deep, triangular ulcer. There was a little ulcerating crinkling in the inter-arytaenoid region. The condition again appeared tubercular.

Improvement continued steadily, and by November 21, 1922, the larynx had advanced to the condition in which it has now remained for five months. No glands in neck; no fresh disease anywhere in the larynx, and the left side remains normal. On the right side the arytaenoid and the inter-arytaenoid area are again normal. The right endo-larynx is quite fixed, as if the vocal cord was adherent to the outer wall. The ventricular band is not infiltrated.

Below it there appears the anterior two-thirds of a fixed white vocal cord, or the cicatricial cord which has replaced the natural one. The present appearances are, indeed, not unlike those left after a successful laryngo-fissure for intrinsic cancer.

The patient does not complain of anything but his weak voice (he is unable to close his glottis completely). His general health is satisfactory, except for his cardiac condition.

The case is extremely puzzling. It may be recalled that his laryngeal trouble started two and a quarter years ago. Drawings made by three independent observers show a decided and extensive lesion. The process has apparently completely cicatrized. If the microscopic specimen is to be thoroughly relied upon, it looks as if retrogression and complete scarring had been effected by the X-ray treatment.

As recorded, the laryngoscopic appearances have at times seemed more tubercular than malignant. (But spontaneous healing, occurring so rapidly in five months and leaving a fixed cord, is unusual in tuberculosis.)

Could it have been luetic in spite of the negative Wassermann? (Specific conditions sometimes heal spontaneously and rapidly.)

The further history will be reported.

A drawing of the larynx before treatment was exhibited, also a microscopical section of a part of the growth.

#### DISCUSSION.

Sir JAMES DUNDAS-GRANT said that tuberculosis had been suspected at the time that he (the speaker) had removed the outgrowth on the vocal cord for examination, but the microscopic section and photomicrograph had shown its typical epitheliomatous nature. The success of Dr. Knox's treatment of this case by X-rays alone had been most brilliant.

Dr. P. WATSON-WILLIAMS asked whether the exhibitor usually removed the ala, and whether any advantage was gained by doing so; also he inquired as to the length of the X-ray séances in the third case.

Mr. A. J. M. WRIGHT asked whether Sir StClair Thomson ever sutured the wound in the larynx. If not, had he any theoretical objection to the practice? In a recent case he (the speaker) had inserted catgut stitches, and had had no reason to regret it.

Dr. ROBERT KNOX said that the third patient had been treated by the ordinary technique employed for several years at King's College and the Cancer Hospitals, except that a larger dose at more frequent intervals had been employed. The radiation was the most penetrating which the hospital apparatus, a 16-in. coil, would yield, increasing up to a 10-in. spark, approximately 130,000 to 150,000 volts. The filtration was through 8 mm. of aluminium, and a pad on the skin provided a second filtration to prevent secondary radiations damaging the skin. Twice a week for a month patient had had a full dose of the rays directed on to his larynx, first from the left side, then from the right, followed for several months at fortnightly intervals. Altogether he had had twenty hours of exposure, spread over a considerable time. A striking feature was that this frequent exposure had produced no effect on the skin. It was a treatment he had used in other cases—e.g., in tuberculous glands—with a favourable response. Similar treatment had been used in another case of sarcoma of the pharyngeal wall, sent to him by Dr. Dan McKenzie eighteen months ago. The same technique had been employed over a long period, and the growth had disappeared. These two cases were the most successful he had had.

Mr. A. J. HUTCHISON referred to six of his patients with intralaryngeal cancer, upon whom Sir StClair Thomson had operated. One operated upon three months ago had had a growth which had extended to the front of the cord, and had probably

crossed the middle line. In this case Sir StClair divided the thyroid cartilage on the opposite side of the middle line, and so far there had been no recurrence, though it was early yet to be certain of the result. The success of these operations had been very striking. Of the six patients he (Mr. Hutchison) had sent to the exhibitor, he had lost sight of one, one had died of pneumonia several years after the operation, one, an alderman, was now able to preside at meetings and to make himself heard in a large room, one was a former policeman, now aged over 80, and the others were well.

Dr. ANDREW WYLIE inquired why Sir StClair Thomson had discontinued the practice of removing a piece of the growth—especially if projecting—for microscopical examination? The omission to do so had resulted in his (the speaker's) hands in one or two mistakes; e.g., in one case he had performed laryngo-fissure and then found that the condition was tuberculous; he had seen two similar cases in the practice of others in which this mistake was also made. It was a simple procedure to remove a piece of growth endolaryngeally. Sir StClair had referred to the free mobility of the cord, which indicated that the cord was not deeply involved by the growth, or there would be a want of movement, not necessarily a fixed cord, but sluggish action.

Dr. WILLIAM HILL said that he also had performed laryngo-fissure in a case of tuberculosis of the larynx; he had found it a good form of treatment where the disease was chronic and limited to one cord, a form which in appearance simulated malignant disease. In such a case there was no indication to remove the thyroid ala.

Mr. E. D. D. DAVIS asked how many cases of laryngeal carcinoma Dr. Knox had treated by X-rays, and in how many had good results been obtained? Some patients came to hospital for the first time suffering from urgent dyspnoea, and requiring tracheotomy; he (Mr. Davis) had had four such cases within three years. Was there any possibility in these cases of a complete removal of the growth by laryngo-fissure? In two of the cases the extent of the growth had been determined by laryngo-fissure, but complete laryngectomy had been necessary because the growth had invaded the lateral wall of the larynx. In the other two, tracheotomy had been performed, after which the patients had refused laryngectomy. His (the speaker's) own experience had been that if there was urgent dyspnoea in a case of carcinoma, it was generally inoperable by laryngo-fissure. If he had another case where tracheotomy was urgent, he would attempt to remove the growth at the time of the tracheotomy.

Mr. MUSGRAVE WOODMAN remarked on Sir StClair Thomson's low mortality in these operations. Sir StClair had operated upon a male patient of his (the speaker's), a fat, florid, somewhat alcoholic man, who had septic tonsils and a septic mouth. Yet he had done extremely well, and had been out of doors at the end of the week. With regard to Dr. Wylie's remarks on endolaryngeal removal of a portion of the growth for microscopical examination, he (Mr. Woodman) would remind Dr. Wylie of the case of the Emperor Frederick of Germany, and the history and treatment recorded by Sir Morell Mackenzie in his publication, "Frederick the Noble." Sir Morell Mackenzie had removed portions of the growth on several occasions and they had been microscopically examined by Virchow and others and pronounced non-malignant. He (Mr. Woodman) considered that the patient had died from lack of application of this successful operation and as a result of too much dependence being placed on the opinion of the pathologists.

Mr. HAROLD KISCH said that the oldest patient upon whom he had performed laryngo-fissure was a male aged 79—now 82. The operation had presented no difficulty and had occupied twenty-five minutes. In this case some delay in the healing of the tracheotomy wound had occurred owing to ossification of the rings of the trachea; otherwise the patient had done well.

Mr. CORTLANDT MACMAHON said that he had spoken on the subject of the training of the voice after operation for intrinsic cancer of the larynx before the Medical Society of London in 1919,<sup>1</sup> when several members now present had heard his remarks. The

<sup>1</sup> Discussion on Sir StClair Thomson's paper, "Intrinsic Cancer of the Larynx." *Trans. Med. Soc. Lond.*, 1919, xlii, p. 122.



line to adopt in the re-education of the voice was contraction of the sterno-thyroid and sterno-hyoid muscle in order to sink the larynx and so to get relaxation of the uninjured cord. The styloglossus, stylohyoid, palatoglossus and palato-pharyngeal muscles, being hypertrophied through over-use, prevented the descent of the larynx. Therefore, in a bad case he (Mr. MacMahon) employed the fingers of his right hand so as to depress the back of the tongue; in mild cases he advised the patient to use a glass tongue depressor. In a few weeks after this procedure a marked descent of the back of the tongue and larynx was produced. In many cases of chronic pharyngitis and laryngitis the appearance of the hypertrophied palato-pharyngeal muscle was like an inverted V, and after treatment like an inverted U. The sternothyroid and sternohyoid muscles became so strong that they sank the larynx and then the voice was deep-pitched and the vibrations were produced with very little effort. One of Sir StClair Thomson's patients whom he (the speaker) had seen that day was better, but still had poor vocal technique, the chief fault being over-breathing, which raised the upper chest and caused general rigidity of the throat. By improving the technique of the production of the voice considerable comfort and increase of tone would be obtained. Underlying the vocal treatment of all forms of throat affections was the physical relaxation of the muscles above the larynx and the acquirement of a considerably lower pitch of the voice.

Dr. KNOX (in reply to Mr. E. D. D. Davis) said that he had not yet treated many cases in the way he had described. He had treated a number of cases of patients suffering from advanced laryngeal carcinoma—with the object of affording some palliation. The case just described was one of the first he had had in which more than a palliative result had been obtained, and he had been stimulated to try it by the reports from Continental centres, in which curative effects were claimed for high voltage radiations administered in large doses. The question of the duration of the dose was always an important one. A single large dose was advocated by some authorities, three to six hours at one sitting, others preferred to give the large dose in parts, of one hour each, on succeeding and alternate days. The latter method appeared to be the preferable one, because it did not exhaust the patient so much, and acted quite as favourably upon the local condition.

Sir STCLAIR THOMSON (in reply) said that the remarks had shown the great advantage of discussing a number of similar cases the same afternoon, as had been suggested that day by Mr. Woodman at the Council meeting. Referring to Dr. Watson-Williams' query, Sir StClair said that he (the speaker) now always removed the thyroid ala, and he was quite satisfied with the procedure. Of sixty cases in which he had performed laryngo-fissure, eight were over 70 years of age, and all recovered from the operation. One had died of cerebral hæmorrhage many years after the operation, and six were still alive and well. Mr. Hutchison had sent him a number of cases, and the fact that six patients had come to him from one town showed that the condition was fairly common. Some cases suspected to be malignant were found, after waiting and watching, to be tubercular. In reply to Dr. Wylie, Sir StClair said that his (the speaker's) records showed that in less than half of his sixty cases mobility of the cord was affected. If one waited for the cord to be fixed, one was waiting for the patient to die. Sir Felix Semon and others maintained that in the majority of cases a fixed cord was characteristic of this condition: but more than half the speaker's cases were operated on long before the growth had infiltrated and fixed the cord. It was this teaching concerning fixation of the cord which retarded early recognition of these cases. The reason he did not remove a piece of growth for microscopical examination before operation was because it was impossible to remove a piece from an embedded growth in a vocal cord without doing so much damage to the cord that one might as well perform laryngo-fissure at once. He always removed a portion for examination, if it could be done satisfactorily. There were only fourteen cases out of his sixty in which he considered it was feasible to remove a portion. When the growth was subglottic and embedded, the portion which presented was simply inflammatory tissue. In fourteen of his cases, two were reported microscopically to be innocent, but he went on with the operation, and the disease was found to be malignant. He had recorded and published every one of his cases. The death-rate was three; one of the patients—a very



disappointing case—died of rupture of the œsophagus, caused by vomiting. He agreed with Mr. E. D. D. Davis that when a patient was found on first examination to have stenosis the case was suitable only for laryngectomy. He (the speaker) pleaded for an early diagnosis. He had made only one mistake in diagnosis, a case not included amongst the sixty now referred to. The patient had a negative Wassermann reaction, lungs and sputum gave negative results in tests for tubercle, and this patient was a hale and hearty policeman. When the larynx was opened, he (the speaker) felt the cord and it was soft, but still he thought it must be malignant. He removed the growth along with the cord, and it turned out to be tubercular. Three months later tubercle bacilli were found in the sputum, and the patient had taken a long time to get well. As to the interesting case "cured" by radium, he (Sir StClair) thought the right attitude was to "wait and see."

### **Tuberculosis of the Larynx cured seven years ago by Silence and Galvano-cautery.**

By Sir STCLAIR THOMSON, M.D.

PATIENT, a clergyman, aged 39, was admitted to Midhurst in February, 1915, with disease in the right lung and tubercle bacilli in his sputum. There was deposit in both ventricular bands, and their edges were ulcerated; both vocal cords were pink and abraded. Although put on silence he did not follow the treatment strictly at first; his larynx became worse; he developed a temperature and had to be kept in bed. Hence, five months after admission the whole larynx was injected; the vocal bands became red and swollen, with ulcerating deposit over each vocal process and posterior half of each cord, invading the edges of the ventricular bands. It required six months' further silence and sanatorium treatment before the larynx was sufficiently quiet to warrant treatment with the galvano-cautery. This was applied three times—December 18, 1915; January 22, 1916; and February 25, 1916. On March 24, 1916 (i.e., thirteen months after admission), he was allowed to talk, and on May 6, 1916, the larynx was quite cicatrized and has remained sound these seven years.

The patient subsequently served with the Army in France as a military chaplain. His health is excellent and he fulfils all his duties actively.

Drawings of the larynx before and after treatment were exhibited.

### **Healed Tuberculosis of Lung and Larynx.**

By Sir STCLAIR THOMSON, M.D.

PATIENT, a female, aged about 35, whose larynx healed by silence and galvano-cautery.

This case required eighteen applications of the cautery between August, 1912, and May, 1917. The larynx has since remained scarred. Patient was shown at a meeting of the Section in June, 1921,<sup>1</sup> as a healed case and it was noted that there was some stenosis and shortness of breath. This increased slowly and required a tracheotomy on October 12, 1922.

Patient can now walk for miles and climb stairs; excellent health; carries on work easily in a post office, wearing a low (Durham) tracheotomy tube with a speaking valve in it.

<sup>1</sup> Drawings of the larynx were exhibited.

Sir JAMES DUNDAS-GRANT said he deprecated using the galvano-cautery in the interarytenoid space, because of the risk of setting up cicatrization, which might contract the glottis. He (the speaker) used it freely in other parts of the larynx. In the case shown of "Healed Tuberculosis of the Lung and Larynx," it was fortunate that the cauterization had been carried out high up in the larynx, and that the cicatricial band which resulted had been well above the level of the vocal cords. In the interarytenoid space treatment a little less searching should be tried, such as trichloroacetic acid, which acted well on tuberculous outgrowths and caused less cicatricial contraction than the galvano-cautery. When he (Sir James Dundas-Grant) had first taken charge of the throat cases at Brompton Hospital, he had expected to find the tuberculous patients all getting progressively worse, but that did not prove to be the case, for large numbers had improved very much, and some got well. Much could be done for these patients by paying attention to modern details of treatment. The "silence" treatment was more important than anything else. Some time ago he (the speaker) had shown before the Section a patient in whose case subglottic infiltration had disappeared with treatment; she had been in a sanatorium, and had been sent away because it was considered that nothing more could be done for her. In her case the greatest benefit was derived by syringing through her nose a solution of eucalyptol 1 part in almond oil 20 parts; this trickled into the larynx, trachea and ventricles. Another plan was to inject by an intralaryngeal syringe a solution of argyrol; he (Sir James) had seen improvement in subglottic infiltration within a week after this had been done. A mixture of amethesin and orthoform inhaled into an œdematous larynx seemed to diminish the degree of œdema, and patients were enthusiastic about the relief from pain afforded by this. Even a cancer patient said he felt a different man after using it in conjunction with Dr. Knox's treatment.

### Case of Epithelioma of the Vestibule of the Nose after Treatment by Radium.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

MRS. R., seen in the throat department of the Cancer Hospital for the first time on July 5, 1922, complaining of soreness and obstruction in the left nostril. A pale warty growth protruded and a piece of it was removed for microscopical examination. It was reported to be epithelioma.

The patient has been continuously under treatment by Dr. Knox with radium and after some months a portion of the stump of the growth was removed for microscopical examination, which showed changes in the nature of the epithelial cells produced by the radium as follows: "Tissue shows an epithelioma, one aspect of which shows a superficial ulceration. The cells of the superficial epithelium are vacuolated and necrotic, and there is a well marked invasion of polymorphonuclear leucocytes, together with small areas of hæmorrhage. The corium shows, in addition to polymorph cells, granulation tissue cells and fibroblasts. The epithelial layers on the side opposite to the ulcerated surface are little affected." A very noticeable feature is the thickening and hyaline degeneration of the walls of the arteries, shown in the second section as resulting from the action of the radium. The effects of this change on the nutrition of the tissues must be very considerable.

Microscopical sections of the growth before and six months after treatment were exhibited.

#### DISCUSSION.

Mr. HAROLD KISCH inquired as to the nature of an ulcer—the scar of which could be seen—that had been removed from the nose by Mr. Bidwell some years ago.

Dr. ROBERT KNOX (in reply) said that apparently the scar was from a rodent ulcer. The present growth had been examined by a pathologist, who had reported it to be an epithelioma. The changes seen in the section were typical of those produced by radium or X-rays, and he (Dr. Knox) did not think it was material which form of radiation was used, provided the dosage was right; the important point being the amount of radiation absorbed. Radium was the more easily applied, as a small tube could be introduced into almost any space, and in the accessory sinuses it could be applied in small tubes or applicators specially constructed for the case. If the affected area was extensive, one bombarded it with the radiation from an X-ray tube in addition to the local application of radium.

### Case of Fibroma of the Nose.

By LESLIE POWELL, M.B., B.C.

PATIENT, a boy, aged 13, had suffered for three months from nasal obstruction, causing mouth-breathing and snoring, also on one occasion slight bleeding. Transillumination showed both maxillary antra clear. A tumour was seen through the right naris, attached posteriorly to the floor and the adjacent parts of the choana. Removal of the growth in March, 1923, was accompanied by free hæmorrhage, which soon ceased.

*Pathologist's Report.*—A pure fibroma.

On March 29 a recurrence on the inner wall was removed.

### DISCUSSION.

Mr. FRANK ROSE asked whether this case had been examined under an anæsthetic, in order to discover the exact point of origin of the growth. The notes of the case suggested that it was attached to and growing from the posterior part of the septum, but that was unusual. He (Mr. Rose) had examined several cases, and his experience was that the growth was attached to the outer wall of the nose, not primarily to the septum.

Mr. LESLIE POWELL (in reply) said that he had removed the original growth under an anæsthetic; it originated from the inside of the naris, not from the nasopharynx, and was attached to the nasal floor and septum. He removed it with scissors, inserting his fingers into the nasopharynx as a guide, since he was unable to see what he was doing. Later he was able to see the position from which it was growing through a small piece left behind, and it was not from the outer wall. He thought he would try to eradicate it by diathermy.

*Postscript.*—A further recurrence, as seen when shown at the meeting of the Section, was removed later, and was found to be attached to the ethmoid region. The nares now appear to be quite clear of growth.

### Foreign Body removed from the Trachea of a Child, aged 6 months.

By H. SMURTHWAITE, M.D.

It was known that the child had swallowed the little round flat whistle which had become detached from her rubber doll, but no notice was taken at the time as she seemed none the worse. Some four weeks later the child's breathing became embarrassed and she was sent to the clinic. An X-ray examination revealed a foreign body at a level with the entrance of the left bronchus. The smallest sized bronchoscope was inserted and the whistle was

plainly seen, but could not be extracted with the forceps at hand because they obstructed the view. However, an improvised instrument was made and the foreign body successfully removed.

Instrument, foreign body and skiagram were exhibited.

#### DISCUSSION.

Dr. IRWIN MOORE said that the successful removal of this foreign body in so young a child demonstrated the principle that Chevalier Jackson had so often emphasized, namely, that in removing foreign bodies the operator should not restrict himself to any one instrument, or method, but should carefully consider the problem of extraction which presented itself, and devise the best means to suit each particular case. He warned members of the danger of using hooks for extracting foreign bodies from the lungs, and referred to an experience which he had shared with Sir StClair Thomson, and recorded by him. During an attempt to loosen, with Killian's hook, a tooth accidentally inhaled and firmly impacted in a secondary bronchus of a child, the hook had caught in a bifurcation beyond the tooth, and all efforts to extricate it had failed. After a considerable time of great suspense and anxiety, the hook had to be torn out of the lung, fortunately causing only slight traumatism, and no after-symptoms, the tooth being later successfully removed by lower bronchoscopy. What might have happened if a vessel in the lung had been torn can be conjectured. Realizing the dangers of such hooks, with curves greater than a right angle (such as Killian's and the one now shown) he (the speaker) had designed a number of probes and hooks which could be safely employed in the lungs, and these had been included in his armamentarium. Another interesting case, not yet recorded, in which he was consulted by a colleague, exemplified the importance of dealing with every foreign body as a mechanical problem. An adult male patient with an old stenosed syphilitic larynx, wearing a tracheotomy tube at night and a vulcanite obturator by day, accidentally aspirated the tracheal portion of the obturator (which had become separated from the neck shield through loosening of the screw) into a secondary bronchus, where it had become firmly impacted, point downwards, presenting to the bronchoscope the circular base, with a diameter greater than the possible opening of any forceps, so that all had failed to grasp it. A long probe was immediately made with a thread corresponding to the screw still attached to the obturator plate, and by passing this probe through the bronchoscope and screwing it into the worn hole in the obturator, it was possible to withdraw the foreign body.

Mr. SMURTHWAITE (in reply) said that he had carefully considered what might happen in the use of a hook in the air or food passages. It would be seen that in the instrument shown the hook was blunt and could hardly do damage to the tissues. He (Mr. Smurthwaite) had practised beforehand with this instrument by removing a foreign body similar to the one in this case placed in a rubber tube.

### **Case showing Results following the Accidental Swallowing of Sulphuric Acid in a patient with Syphilitic Laryngitis.**

By C. GILL-CAREY.

MALE, aged 56. When first seen, in July, 1922, he complained of hoarseness of six months' duration. With some difficulty a view of the larynx was obtained. The greater part of the right cord was occupied by a deep ulcer with sharply defined edges; on the posterior third of the left cord there was a small smooth swelling. Wassermann reaction strongly positive. In October, 1922, while under treatment for his laryngeal condition, he accidentally swallowed some sulphuric acid. Within a month of the accident he began to

experience difficulty in swallowing and when seen again he could only swallow fluids. Œsophagoscopy showed scarring of the hypopharynx and a fibrous diaphragm occluding the right half of the œsophagus about 2 cm. below the cricoid. Dilatation was effected through the œsophagoscope. Improvement in swallowing followed but subsequent dilatation by the passage of bougies was unsatisfactory owing to the frequent impaction of the tip in a cul-de-sac at the side of the stricture. Dilatation under vision, was therefore produced on several occasions, with some improvement. In spite of energetic anti-syphilitic treatment, including six injections of arseno-benzol, very little change took place in the larynx. Moderate stridor and dyspnoea on exertion were always present.

On February 24, 1923, œdema of the glottis followed the passage of a bougie and a tracheotomy had to be performed, and since then the ulceration has been steadily healing, but the airway is poor. Large bougies can now be passed through the œsophageal stricture, but there is still moderate dysphagia. Suggestions as to further treatment of the larynx and œsophagus are invited.

Dr. WYLIE suggested the local application of a solution of copper sulphate so as to reduce the cicatricial tissue in the larynx. He did not think it was possible to dispense with the tracheotomy tube.

### **Case of Swelling in the Nasopharynx on the Right Side, displacing the Soft Palate downwards.**

By W. M. MOLLISON, M.Ch.

PATIENT, a female, aged 23. Pain in the right ear commenced a year ago, for which, two months later, she was treated at Golden Square Hospital. A month later the pain spread to the right side of the face. Removal of adenoids four months afterwards relieved the pain in the ear for a time, but the pain in the face remained. The patient was first seen at Guy's Hospital, by exhibitor, in March, 1923, still complaining of severe pain in the right side of the face and in the right ear, and also of some deafness. The swelling in the nasopharynx was then observed. Wassermann reaction positive. A portion of the growth removed was examined microscopically and reported to be sarcomatous.

#### **DISCUSSION.**

Mr. T. B. LAYTON (in the absence of Mr. Mollison) said that advice was asked as to what should be done. Sir Charters Symonds had seen the case, and advised that the pathologist's report should be ignored and the case treated as one of syphilis.

Sir JAMES DUNDAS-GRANT said that the enlarged gland made the case appear worse than it really was; he did not think that enlargement was connected with the disease in the nasopharynx, but believed it was caused by a septic tonsil, which should be enucleated.

### **Two Cases of Paralysis of the Left Vocal Cord and Left Half of the Palate.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

Case I.—Patient, a male, aged 32, suffering from neurasthenia and attending at the West End Hospital for Nervous Diseases, developed hoarseness about a month ago. Examination revealed paralysis of the left vocal

cord and left half of the palate. Inquiry elicited the history of a primary syphilitic lesion a year ago, followed by eruptions, and he is being treated for the specific disease at a special hospital. The laryngeal condition is in all probability an early syphilitic involvement of the vagus, above the region of the pharyngeal branch. There is slight facial paralysis on the right (opposite) side. Taste (glossopharyngeal and chorda) is impaired on the right side, but normal on the left. These conditions on the right side are due to suppuration in the right middle ear.

*Case II.*—Patient, a male, aged 46, recently seen at the Cancer Hospital, suffering from dysphagia especially for liquids. There is almost complete immobility of the left vocal cord and there is paresis of the left half of the palate: the glossopharyngeal taste is deficient on the left side. The larynx is otherwise normal. Wassermann reaction positive. Since taking mercury and iodide of potassium for a week the patient has felt better and has less difficulty in swallowing.

### **Outgrowth from the Anterior Half of the Left Vocal Cord ; ? Fibroma or Prolapse.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a female, aged 40, was first seen at Brompton Hospital a month ago suffering from hoarseness, with a suspicion of tuberculosis. A smooth rounded growth was seen projecting from the anterior half of the left ventricle, a portion of which was removed with forceps and microscopically reported to consist of richly cellular structure with numerous small round cells, having the appearance of a granuloma, without any evidence of tuberculosis. No physical signs of tuberculosis were present and no bacilli were found in the sputum, even after a cough was induced by the sniffing of mustard oil.

Dr. IRWIN MOORE said that he considered the condition in this case was a hyperplastic fold of mucosa protruding from the sacculus laryngis, wrongly described by some observers as "prolapse of the ventricle."

### **Case of Sarcoma of the Tonsillar Region treated by X-rays after Partial Removal.**

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D., and  
DAN MCKENZIE, M.D.

PATIENT, a male, aged 67, had suffered from throat trouble since September, 1920: partial removal of a sarcomatous growth was effected in November and December, 1920, by Dr. Dan McKenzie, who referred him for X-ray treatment to Dr. Robert Knox. This treatment was commenced in September, 1921. In the beginning of 1922 a pale irregular swelling was observed below the right tonsil, but in May there remained little except pale firm fibrous tissue. When first seen the right cord was paralysed. In May, 1922, the movements of the larynx were normal. In November, 1922, an erythematous patch with circinate edge was observed on the right half of the soft palate, which was probably due to irritation caused by smoking, as it disappeared when the patient discontinued the habit.



Dr. R. KNOX said he had already alluded to this case. The original growth was in the pharynx, and the tonsillar condition was a recrudescence. Dr. McKenzie had operated twice upon the case, and the microscopic examination confirmed the diagnosis of sarcoma. The patient had been under treatment eighteen months, and the growth had disappeared under X-ray treatment. The patient was attending the Cancer Hospital for X-ray treatment and was under the joint observation of Sir James Dundas-Grant and himself. Sarcoma was more responsive to X-rays than were other growths.

### **Specimen of a Large Cyst of the Orifice of the Larynx arising from the Arytæno-epiglottidean Fold.**

Exhibited by E. D. D. DAVIS, F.R.C.S.<sup>1</sup>

REPORT BY S. G. SHATTOCK, F.R.S.

A LARYNX opened up from behind. Projecting into it close above the ventricular band of the right side, there is a spherical cyst about the size of a small cherry, which is prolonged by a constricted process through the thyro-hyoid membrane, beyond which a larger process lies in front of the right cornu of the hyoid bone and the thyro-hyoid ligament. There is no communication between the sacculus or ventricle and the cyst, which possibly arose in the glands of the former, and, after projecting into the larynx, extended extralaryngeally through the thyro-hyoid membrane at the site of a natural nerve or vascular perforation. In the upper part of the thyro-hyoid ligament there is an elongated neck-like process of cartilage which is discontinuous below with the thyroid. The angle of the right ala from which the superior cornu normally arises is smooth and rounded: the condition probably represents a malformation rather than the result of traumatism. The cyst is furnished internally with an epithelial lining.

The specimen has been presented to the Museum of the Royal College of Surgeons.

### **Ulceration of the Left Tonsil: Case for Diagnosis.<sup>2</sup>**

By H. BELL TAWSE, F.R.C.S.

MALE, aged 25. About eleven months ago the mouth became tender; small ulcers appeared on lips, with a larger one on the dorsum of the tongue, and persisted for three months. Some carious teeth were removed, the assumption being that they were the cause of the ulcers.

Six weeks later the lips and tongue had healed but soon afterwards an ulcer developed on the left tonsil and was present when first seen by exhibitor on June 28. The ulcer involved practically the whole tonsil, spreading on to the tongue, and it had a punched-out appearance, the floor being covered with a yellow slough. On the middle of the dorsum of the tongue was a small shallow painful ulcer. No laryngeal lesions were present. A papular eruption mixed up with acne pustules was scattered all over the trunk and limbs, but this had been present for about two months.

<sup>1</sup> At a meeting of the Section on March 2. See *Proceedings*, 1923, xvi, p. 54.

<sup>2</sup> This case was shown at the meeting of the Section held November 3, 1922. Publication of notes and discussion was deferred pending further investigations.



Tertiary syphilis was diagnosed and potassium iodide, 15 gr. t.d.s., was prescribed. Three Wassermann tests proved negative. No improvement in fourteen days. Mercurial inunction and novarseno-benzol injections were next tried and potassium iodide was discontinued. Fourteen days later the tonsillar ulceration was rather worse and numerous ulcers had appeared on the gums. Inunctions discontinued, potassium iodide again prescribed, 30 gr. t.d.s.: novarseno-benzol injections continued. Wassermann test still negative. No tubercle bacilli in sputum after repeated examinations. No lesions in the lungs could be found, nor Vincent's spirillum.

Section of piece of tonsil removed shows only chronic inflammatory changes with a good deal of fibrosis and some infiltration of the mucous membrane with small round cells. Actinomyces not found.

Swabs from ulcers on tongue and tonsil, as well as cultures, showed *Pneumococcus mucosus*, *Micrococcus catarrhalis*, and various streptococci. Glanders bacillus negative.

The dose of potassium iodide was now increased to 60 gr. t.d.s., and in three weeks the ulcer on the tonsil was almost healed. During this time the novarseno-benzol injections had been continued. Three weeks later ulceration had returned and had spread downwards, involving the entire epiglottis, which was swollen, cedematous and sloughing. The whole ulcerated area was freely cauterized with the electro-cautery, the potassium iodide was continued and the patient was sent to the seaside. In a month's time the epiglottis was healed and the tonsil was almost healed.

The patient was well until a fortnight ago, when the ulceration of the tonsil recurred.

Two injections of autogenous vaccine (detoxicated) prepared from throat swabs were given at intervals of one week, on the last occasion followed by very marked local reaction with abscess formation. The vaccine was discontinued. Now X-rays are being applied to the left tonsillar region, and potassium iodide—30 gr. t.d.s.—is being given.

#### DISCUSSION.

Mr. HERBERT TILLEY said that he regarded the condition as a form of tuberculosis.

Sir JAMES DUNDAS-GRANT said he thought the case was tuberculous. He had had a case in which one half only of the larynx had been involved, suggesting epithelioma, but he made a diagnosis of tuberculosis. The patient had disappeared, and, ultimately, had been sent to the Cancer Hospital. He (the speaker) had again seen the patient, and had removed a portion of tissue from the left side of the epiglottis, and found that it contained giant cells, although there had been no pulmonary signs, and no tubercle bacilli had been found in the sputum.

Mr. BELL TAWSE (in reply) said he regarded the condition as a mixed infection of tubercle and syphilis. He would report further.

December 1, 1922: Patient was again shown to the members, as there had been a new development in the case. A large ulcer, covered with a whitish slough, had appeared on the anterior pillar of the fauces, and was spreading on to the adjacent part of the palate.

#### DISCUSSION.

Sir JAMES DUNDAS-GRANT said that the character of the ulceration and its situation recalled the self-inflicted injuries occasionally encountered during the war.

Mr. C. A. PARKER (President) said that the ulceration had extended enormously since last meeting and the epiglottis was now considerably involved. It was an

interesting case and presented considerable difficulty from the diagnostic point of view.

Mr. J. F. O'MALLEY asked whether the extension of the ulceration was spontaneous, or was a result of a portion having been removed for sectioning?

Mr. BELL TAWSE (in reply) said that the ulceration on the pillar was spontaneous. The sectioned portion of the epiglottis and tonsil did not help the diagnosis. He believed it to be tubercular.

May 2, 1923: The day after the meeting, that is, on December 2, the patient caught cold and became very ill, with a temperature of 104° F.: the palate was fiery red, yellow spots appeared on the swollen right tonsil, the sloughing area on the left pillar now involved the tonsil and extended rapidly upwards and to the middle line affecting the uvula and much of the left palate. Ulcers appeared on the tongue, gums and lips coated with whitish deposit, the larynx was red and swollen and some dyspnoea was present relieved by inhalations. On the suggestion of Mr. Mark Hovell, 5 c.c. colossal argentinum was given hypodermically, a fall of the temperature to 102° F. followed, and the patient was more comfortable. Twenty-four hours later another 5 c.c. were given and the ulcers were freely painted with the same preparation and the temperature fell to 99° F. Subsequent injections were given every four days until a crop of boils appeared, when they were discontinued. By this time the ulcers on the lips and tongue had healed and all the swelling in the larynx had disappeared, but the ulcer on the left pillar and palate, though smaller in size and cleaner, remained much as before. The left tonsillar ulcer had healed and the epiglottis looked clean and healthy. On January 1, 1923, the temperature again rose to 100° F. and the patient developed an acute right follicular tonsillitis. Injections of 2 c.c. colossal argentinum were resumed and in forty-eight hours the temperature was normal. It rose again for twenty-four hours, then remained normal for six days. A further twenty-four hours' rise of temperature to 100° F. was followed by several weeks of normal temperature. Injections were discontinued on January 20. As the ulcer on the left pillar and palate persisted—neither improving nor getting worse—he was sent to the Radium Institute for radium treatment. X-rays showed old but quiescent tubercular disease in the bronchial glands and lungs, and the opinion was held that the epiglottis was tubercular. Being greatly perplexed as to further treatment I sent the patient into the country to live in the open air, to eat as much as he could, and to take cod-liver oil. In six weeks the ulcer had completely healed and there was no active lesion in the mouth, pharynx or larynx. The patient has remained well ever since. The diagnosis, therefore, remains undecided by all laboratory tests, but I should say that clinically, the case was one of tubercular ulceration of the left tonsil, palate and epiglottis.

## Section of Laryngology.

President—Mr. CHARLES A. PARKER, F.R.C.S.Ed.

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### FIFTH ANNUAL SUMMER CONGRESS.<sup>1</sup>

(Held at Manchester, June 15 and 16, 1923.)

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#### Chronic Hyperplasia of the Upper Jaw: its Relationship to other Osseous Tumours and to Otosclerosis.

By DOUGLAS GUTHRIE, M.D.

(ABSTRACT.)

THIS rare disease appears to deserve greater attention than it has hitherto received. It consists of a unilateral enlargement of the upper jaw, due to exuberant growth of cancellous bone. The maxillary antrum is much reduced in size. Two cases have come under the writer's notice, one in a boy, aged 5, the other in a girl, aged 16. In neither subject were carious teeth present.

Christopher Heath devotes a chapter in his book on "Diseases of the Jaws" to hyperostosis and mentions the case of a woman, aged 25, whose jaw had been the site of a painless enlargement for ten years. The specimen from this patient is in the Royal College of Surgeons' Museum. In the same museum there is a maxilla, excised by Lord Lister, bisected to show the nature of the growth. A specimen in the Museum of Charing Cross Hospital is figured in Colyer's "Dental Surgery and Pathology."<sup>2</sup> This disease must not be confused with leontiasis ossia, a name intended by Virchow to denote multiple exostoses of the bones of the face and head. [Lantern slides illustrating early and advanced cases of leontiasis (Bickersteth, Horsley, Bland-Sutton) were shown.]

The strange anomaly of unilateral hypertrophy, described in detail by D. M. Greig, is an enlargement of normal tissues and not a hyperplasia.

On microscopic examination, chronic hyperplasia of the upper jaw presents an appearance very similar to that of otosclerosis. In the writer's cases, Dr. Dawson described a network of new bone, whose meshes, irregular in size and form are filled with a loose fibrous tissue. At the periphery, closely applied to

<sup>1</sup> The papers, unless otherwise stated, will be published *in extenso* in the *Journal of Laryngology and Otology*.

<sup>2</sup> Colyer, "Dental Surgery and Pathology," London 1923, p. 842.

the bone are several layers of osteoblasts, engaged in depositing new bone. Very few osteoclasts, and only a few scattered lymphocytes are present. No deafness has been noted in any of the recorded cases but the histological similarity is interesting and analogous to that described by Mr. G. J. Jenkins in his recent paper on "Otosclerosis and Osteitis Deformans."<sup>1</sup>

#### DISCUSSION.

Mr. LAYTON said that this disease occurred in the chimpanzee. He considered it was a very interesting subject and hoped Dr. Guthrie would continue his investigations. In his opinion the causes of this condition of hypertrophy of the upper jaw particularly required investigation. He would like to hear Dr. Guthrie's views as to the possibility of some microbic infection being the original cause.

Mr. F. H. WESTMACOTT said he had been interested in these cases for a considerable time, and thought the point which required special investigation in the clinical aspects of these cases—in differentiation from other diseases of the bone of the superior maxilla—was the swelling in the canine fossa; in other diseases it encroached upon the orbit and the nose. He (the speaker) had not seen any case which was not limited by the orbital plate, it never invaded the internal wall. It always affected the antrum.

Dr. GUTHRIE (in reply) said that with regard to Mr. Layton's suggestion of microbic infection causing the hyperplasia, the microscopic appearances gave no support to such a view of the aetiology. As to the question of carious teeth—the study of literature would rather suggest that dental caries was rare, and in the specimens which he showed there was no caries. Regarding Mr. Layton's reference to nodular growth in the upper jaw of a chimpanzee he (Dr. Guthrie) thought this was probably a case of "osteitis fibrosa."<sup>2</sup> Mr. Westmacott's suggestion as to the transition from this disease to sarcoma was very interesting. In the case of the kidney the possibility of the transition between adenoma and sarcoma had been raised by Mr. John Fraser, of Edinburgh.

### Some Clinical Observations on the Lingual Tonsil.

By T. ARNOLD-JONES, O.B.E., F.R.C.S.Ed.

(ABSTRACT.)<sup>3</sup>

OBSERVATIONS based on fifty-three cases—forty-nine private, four hospital—seen from the latter half of 1919 to the end of 1922. The region of the lingual tonsil is largely responsible for certain symptoms which may be divided into three groups:—

(1) Thirty-four cases with vague aches and pains, discomforts and sensations in the throat, come within this group.

(2) Twelve cases with paroxysmal cough, often most intense and persistent, distressing alike to patient and friends. In all these cases disease of the lungs was definitely excluded.

(3) Seven cases with dysphagia varying from slight difficulty in swallowing to absolute inability to take solid food, come under this heading.

<sup>1</sup> *Journ. Laryngol. and Otol.*, 1923, xxx, p. 144.

<sup>2</sup> (See paper, by R. Lawford Knaggs, *British Journal of Surgery*, x, p. 487.)

<sup>3</sup> This paper is published in *extenso* in the *Journal of Laryngology and Otology*, 1923, xxxviii, p. 212.

The appearance of the lingual tonsil in these cases varies from the normal to that of definite hypertrophy and varix. In twenty-four cases out of fifty-three no departure from the normal was noticed.

*Treatment.*—(1) Local: Applications of a 1 in 40 solution of silver nitrate to the region of the lingual tonsil; galvano-cautery to the same region; the daily use of an oily spray containing menthol. (2) General: Correct any alimentary or intestinal disturbance; treat gout or rheumatism; combat neurosis with bromides and valerian; treat endocrine insufficiency with appropriate glandular extracts.

*Results of Treatment.*—Ascertained by forty-one replies from fifty-three patients written to. Results divided into four groups:—

(1) *Cured*: eleven cases. The patient declared himself absolutely free from all symptoms, and had remained free for a minimum period of six months.

(2) *Much improved*: eleven cases. The patient says he is much better but has occasional relapses.

(3) *Improved*: fifteen cases. Definite improvement has taken place but the symptoms still persist in minor degree.

(4) *No better*: four cases.

Taking the cases in their groups according to symptoms the effects of treatment are as follows:—

*Group 1.*—Comprising the vague aches and sensations. Thirty-four cases: six cured, four much improved, twelve improved, three no better, and eight no reply.

*Group 2.*—Paroxysmal cough. Twelve cases: three cured, four much improved, two improved, one no better, one no reply.

*Group 3.*—Dysphagia. Seven cases, two cured, three much improved, one improved, one no reply. The two patients who were cured complained of absolute inability to take solid food.

#### DISCUSSION.

Mr. C. A. PARKER (President) stated that at one time he had made a good many observations as to the possible connexion between abnormalities of the lingual tonsils and the various symptoms sometimes attributed to them, and he had come to the conclusion that on the one hand there might be considerable changes in the tonsils without any symptoms whatever, and on the other hand there might be many symptoms without any changes being present. It was perfectly true that relief of symptoms might be obtained by cauterization of the lingual tonsils, but the same relief followed cauterization of the posterior wall or lateral bands of the pharynx, or the application of any form of counter irritation to any part of the pharynx. He did not think the lingual tonsils were responsible for the symptoms described.

Dr. WILLIAM HILL said that having seen a number of these cases with X-ray reports, he had never found bismuth remaining behind in the deep pharynx, a fact which showed that there was no obstruction to swallowing. Mr. Jones had exhibited X-ray plates with bismuth in the deep pharynx, and although he would not say his own researches were exhaustive, Mr. Jones had found what he (the speaker) had not discovered. In his (Dr. Hill's) opinion it was not advisable to rely too much upon the radiographer's report on the œsophagus.

Mr. M. VLASTO said that a good many of the cases which Dr. Jones had reported had probably no organic basis. The term "dysphagia" was used indiscriminately to denote difficulty or pain on swallowing. This had often given rise to misunderstanding. He attributed more importance—as an expression of local affection of the faucial region—to pain produced by a dry swallow than to that produced by the swallowing of food.

Dr. P. WATSON-WILLIAMS said that in his experience the lingual tonsil was very seldom responsible for pathological symptoms. Of course it did participate in inflammatory conditions of the throat, and it might be the seat of rheumatic or septic infection, abscess, &c., but such cases were few and far between. He had seen cases of most extraordinary hypertrophy of the lingual tonsil, and as in these there had been an incessant cough from which the patient had failed to get relief he had felt it necessary to operate and remove the hypertrophy. He did not think he had cauterized the lingual tonsil for twelve years at least, though many years previously he had himself frequently attributed symptoms to this lingual tonsil, and used the cautery, as he now believed, unnecessarily. He took this opportunity of explaining definitely why he could not regard the lingual tonsil as being the essential cause of all the symptoms described by Dr. Arnold Jones.

Sir WILLIAM MILLIGAN said that he had taken a particular interest in the lingual tonsil. Although he was indebted to Mr. Jones for raising the point, which was worth discussing, he (Sir William) would say at once that during the whole of his professional career he had never been able to associate more than one or two cases of enlarged lingual tonsils with any definite clinical condition. He thought the symptoms described were purely nervous. They occurred, as a rule, in highly excitable women as a local expression of a general nervous condition. He thought there was considerable risk in surgical interference with the lingual tonsil; cases had been recorded in which very severe and dangerous hæmorrhage had occurred. At the same time one would take risks if one could definitely associate clinical symptoms with pathological conditions. So far as his experience went, the lingual tonsil was a very harmless organ.

Mr. TILLEY said that he also thought that the symptoms described as the result of enlargement of the lingual tonsil were of nervous origin. Mr. Lennox Browne—the chief upholder of the significance of varicose veins at the base of the tongue—mentioned spasmodic wry-neck and paralysis of the deltoid as symptoms of that condition. He (Mr. Tilley) had seen several cases in which cauterization of the lingual tonsil had failed to cure local discomforts, and he had come to the conclusion that any improvement was due to imagination. That opinion was expressed twenty-five years ago. Probably several members present would recall the very sharp controversy which occurred about that time. He (the speaker) could not bring himself to believe that so many laryngologists could possibly have overlooked the significance of this region had there been truth in the theory. It was quite uncommon to find these symptoms in men, whereas they were frequently present in women who led sedentary or unhealthy lives, and more particularly at the time of the menopause. He thought these facts pointed to the symptoms being of nervous origin, rather than due to any local lesion in the lingual tonsil. As to the relief obtained from cauterization or local applications of pigments, he thought patients would get equal relief if the application were to the back of the neck.

Mr. H. J. BANKS-DAVIS said that the majority of students nowadays seemed under the impression that it was useless to enucleate the tonsil with the guillotine unless the lingual prolongation was enucleated at the same time, and they were taught that unless the tonsil were removed by dissection the patients would not get the relief required. This was perfectly untrue. He was under the impression that the lingual tonsil was a mucous gland, and that its function differed from that of the faucial tonsil in consequence.

Mr. W. S. SYME said he agreed that one did not generally look to the lingual tonsil as a cause in cases of difficulty of swallowing, &c. At the same time he was quite convinced that he had seen a few cases in which an enlarged lingual tonsil had caused symptoms of spasmodic cough and difficulty in swallowing, and in which the removal of the enlargement had resulted in relief of the symptoms.

Mr. ARNOLD-JONES (in reply) said he was glad that his paper had, at any rate, brought about a discussion. He believed he had been misunderstood, because he did not contend that it was necessarily the presence of an enlarged lingual tonsil which



caused the symptoms, but that it was something which occurred in that area analogous to what occurred in the sensitive areas of the nose in spasmodic rhinitis. In none of his cases had he met with sufficient hypertrophy of the lingual tonsil to justify removal. He maintained that cauterization of this lingual tonsil area undoubtedly brought relief, and if such a simple measure could produce absolute comfort in cases of spasmodic cough, and in some cases of dysphagia, he did not see why it should not be more widely used. The position he had taken up in his paper was that this area had been overlooked. He thought that in these days of high grade surgery a small region like this, which offered no opportunities for heroic operations, was likely to escape notice. He (Mr. Jones) thought it called for more attention than it apparently received at the present time.

### **The Treatment of Large Foreign Bodies impacted in the Gullet.**

By D. R. PATERSON, M.D.

(ABSTRACT.)

No problem gives the endoscopist more concern than that of dealing with foreign bodies in the upper food passages, a problem arising, not infrequently, from the size or irregular form of the object.

Large bodies are usually impacted in the upper segment of the gullet, above the level of the suprasternal notch; more rarely, about the hiatal opening. Besides being bulky, they may be jagged in form, with edges and points which impinge on the wall of the tube and constitute a danger from penetration and infection of the surrounding tissues, demanding prompt and efficient measures.

Among the commoner objects met with are dentures, pieces of bone, metal discs, pins of various forms, &c. Some, from their mere bulk, become firmly held up, and, if allowed to remain, soon become surrounded by swelling and œdema of the gullet wall, which adds greatly to the difficulty and danger of extraction. Even a large tooth-plate with a smooth surface may be so wedged in that it is hard to move, especially if attempts have been made to push it down with a bougie. It is, however, much more serious when the body has sharp angles and metal hooks which are apt to embed themselves in the mucosa and anchor it fast. A fractured denture of thin vulcanite is always a source of anxiety. Even more dangerous are large pieces of bone with sharp edges and irregular surfaces and crevices which harbour septic material—a factor which increases the risk of pressure ulceration. A patient came into hospital suffering from purulent mediastinitis, four days after swallowing a spicule of bone pointed at each end. Though it had not been interfered with, it was found at the post-mortem that one point had completely perforated the gullet, and the other nearly so. Cracked dentures which have been worn for a time, and badly kept, are liable to set up septic trouble. Safety-pins and pins variously mounted, also present difficult problems.

Before dealing with a foreign body, a careful survey of the case is absolutely necessary. The possession of a duplicate of the object is a real help. Radiograms made in different planes—antero-posterior, lateral and oblique—should be available, so that the exact presentation of the body may be made out and the problem of its extraction considered beforehand. Under general anæsthesia a careful inspection should be made for the purpose of orientation. Tubes of various calibres should be used; the larger give wide access; with the smaller,



close inspection of any particular part of the wall is possible; the expanding tube is often of the greatest service. Attention should be directed to the situation of any sharp point, since to prevent such points from injuring the soft parts during extraction is the crux of the problem. It may be possible by seizing the point to rotate the body, so that the point may be brought into line with the long axis of the gullet and traction made without much fear of consequences. A large smooth body is often difficult to grasp; a long-handled hook passed beyond the body may serve to loosen and rotate it. It is occasionally possible to break up the impacted object and remove it piecemeal, a procedure, however, requiring the utmost care.

Early extraction is of especial importance when the body is of jagged shape and has potential pressure points. Though a smooth body may remain for a long period in the gullet without doing much harm, provided it permits food to pass, an irregular one may rapidly set up septic infection. The variability of septic processes which occur is perhaps to be explained by the difference in nature and degree of virulence of the micro-organisms present. One pointed body may lie *in situ* some days, with little or no local reaction around it; another may be accompanied, in a short time, with a characteristic foetid odour which connotes ulceration. Thus, in the case of a soldier who swallowed a metal disc with a sharp edge and a diameter of half-a-crown, and who was seen on the sixth day after impaction, the odour was a warning to proceed with caution. A large sized tube enabled the body to be carefully loosened and extracted from a deep ulcer on each side of the oesophagus and the patient made a good recovery. On the other hand, a fractured vulcanite tooth-plate, swallowed by a young man, produced four days later a distinct foetid odour, due to an ulcer produced by the pressure of a sharp angle on the left side of the wall of the gullet. A cautious attempt to disengage the plate was made but external oesophagotomy was considered advisable. After exposing the gullet and feeling carefully for the foreign body, I felt the wall break under my finger like a piece of wet paper, and the point presented. Through this rent the broken plate was easily extracted and the patient ultimately did well. This was an instance of deep ulceration quickly produced by a fractured surface already impregnated with septic material.

The onset of septic infection may be merely a matter of hours. A patient suffering from dementia paralytica had swallowed his denture. The occurrence was not noticed until about six hours after, when he was observed to be unwell, with a temperature of 102° F. I saw him about ten hours after the event. A radiogram showed the plate with two sharp clasping hooks and serrated edge impacted partly in the hypopharynx and partly in the gullet. On examination the plate was seen to be very firmly fixed by a hook embedded in the lower part of the pyriform sinus, from which an area of oedema extended on to the side of the larynx. The hook was disengaged without difficulty and protected during the further manipulation which delivered the body. The patient died a week later, and at the post-mortem a septic slough, giving practically a pure culture of streptococcus, was found in the pyriform sinus which the hook had penetrated; this had given rise to aspiration pneumonia. Sepsis may thus constitute a grave factor and these examples emphasize its important bearing on the form of treatment to be applied.

When there is difficulty in extraction, ought we to continue with our endoscopic procedures or to do an external operation?

In the first place, external operation is practically limited to bodies in the upper segment of the gullet. I do not say *entirely* limited, for it is possible

that extraction of a body lying below the suprasternal notch might be greatly facilitated by the closer access obtained through an œsophagotomy wound, in much the same way that a low bronchoscopy through a tracheotomy wound is sometimes an enormous advantage.

When perforation is suspected or actually present, an immediate external operation is indicated. Septic conditions which may favour perforation do not necessarily contra-indicate endoscopic manipulation, though they inculcate caution and impose a limit. In such cases œsophagotomy may be advisable. Figures taken from the statistics of general surgeons show a mortality ranging from 12 to 20 per cent. The form of the neck may have an influence upon the mortality, a long thin neck lending itself to more ready access and better drainage than a short, thick muscular one. Since the introduction of endoscopy, the operation is not often done by laryngologists, but there are occasions on which it is required, and it is desirable to have its indications more clearly defined.

#### DISCUSSION.

Sir WILLIAM MILLIGAN said that the removal of some foreign bodies presented extraordinary difficulties, and he considered that if the foreign body was in the respiratory passage there was every reason for urgency. If it was in the œsophagus, however, one could take one's time, within reasonable limits. He was glad to hear Dr. Paterson lay stress upon the value of dilating the œsophagus during examination. He thought this was of enormous importance, and added materially to the ease and safety with which one could remove the foreign body. There were some foreign bodies, for instance safety-pins, which were difficult to remove, since they were liable to tear the walls of the œsophagus. If open and with the point upwards, a problem presented itself which demanded careful consideration. The question arose as to whether it would not be better, after having located the pin by means of X-rays and also by the use of the œsophagoscope, to perform an external operation at once. Everyone who had had experience of these cases knew how difficult it was to prevent the point of the pin lacerating the wall, and once the wall was lacerated—even in a minute abrasion—there was the risk of septic infection. Therefore, he (Sir William) was of opinion that endoscopists were neglecting to some extent the value of the external operation. It was true that they could remove *almost* every foreign body per *vias naturales*, but not every foreign body could be so removed. In these exceptional cases, although they might be successful in removing the foreign body, they certainly subjected their patients to a great amount of risk in their attempts.

Sir William showed a photograph of a laceration of the œsophagus caused by a sharp pointed bone—which had been impacted in the œsophagus for several days. The patient was vomiting small quantities of blood, and before anything could be done, a severe hæmorrhage occurred and death followed. He also referred to the case of a girl who was said to have vomited a pint of blood the day before she was seen. There was a history that she had swallowed a foreign body (a bone) and on examination a small granulation was seen on the posterior wall of the œsophagus opposite the arch of the aorta. He (the speaker) had decided to leave things alone, and the patient had been put back to bed. The following day patient had another violent hæmorrhage and died, and on autopsy a bone had been found immediately under the granulation, having perforated the aorta. In that case there had been unnecessary delay in sending the patient to hospital. He (Sir William) thought that all foreign bodies which were jagged, pointed and sharp in any way, should be removed immediately, simply because the movement of the œsophagus (if it did not actually perforate) tended to produce laceration and perforation. Another important point was that in many cases the foreign body (for instance a large piece of bone) so filled the œsophagus that one could see no lumen at all. Under those conditions he (the speaker) thought they ought to make more use of hooks—not only one hook, but two hooks—one on either side of the foreign body so as to get it sufficiently dislodged to be able to pass a pair of forceps round or over it. He exhibited a collection of foreign bodies which he had successfully removed.

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Dr. WILLIAM HILL said that most foreign bodies with jagged points were not in the gullet at all, but in the deep pharynx, and the sooner they were removed the better. One must never forget that these foreign bodies perforated very quickly, and it might be advisable to resort to the external operation. Familiarity with this operation made one more ready to embark upon it, and he (Dr. Hill) thought this was a much safer plan than devoting, say, three-quarters of an hour to trying to turn a foreign body with a hook. He agreed with Sir William Milligan that with the constant movement of the gullet the jagged point tended to go deeper and deeper into the tissues and to produce mediastinitis which was nearly always fatal.

Mr. T. B. LAYTON, referring to Dr. Paterson's remarks on the importance of careful observation before attempting to remove a foreign body, asked how long Dr. Paterson thought it was justifiable to wait before attempting removal.

Mr. F. H. WESTMACOTT said that in a large number of cases endoscopists were called upon to remove small dentures—like the specimen which had been handed round—from the lower œsophagus. He (the speaker) thought they should institute a propaganda against the manufacture of these small dentures, as they were the cause of a considerable amount of trouble and danger. Dentures ought to be made to cover completely the whole of the palate. With regard to Mr. Layton's question as to whether one ought to delay the removal of a foreign body or remove it at once, the two sides of the question had already been summarized by Sir William Milligan. He (Mr. Westmacott) thought that in all cases one ought not to neglect the call to remove a foreign body, even although it might not be found in the same situation on arriving. Copper coins ought to be attacked immediately, as in a few hours they corroded the wall of the œsophagus.

Mr. W. G. HOWARTH said that in his experience large solid pieces of bone were the most dangerous foreign bodies because of their jagged nature, and also because they were extraordinarily septic, and it was in cases of this nature that the possibility of external operation was most urgent. The external operation was comparatively easy in the upper part of the food passage, but what was to be done when the body was impacted at the level of the aorta? Was one to approach it from the back, or was it better to adopt the anterior method by taking out a rib and displacing the heart? There were advantages in both methods: certainly the frontal one gave a very good view, and drainage could be satisfactorily arranged for.

Mr. E. MUSGRAVE WOODMAN said he agreed with Sir William Milligan and Dr. Hill on the question of external operation. The operation which Mr. Howarth had mentioned was comparatively easy, but it was not free from danger. The œsophageal wall was composed of soft tissue and had no serous coat.

Mr. H. SMURTHWAITE said that in the case of a child said to have swallowed a foreign body, the statement of the mother should not always be accepted. He (the speaker) had shown a case at the last meeting of the Section of a child aged 6 months who had swallowed the whistle from a rubber toy, and had been seen by a surgeon three weeks previously. The child developed difficult breathing, and was sent into hospital. He (Mr. Smurthwaite) had located the foreign body lying at the level of the tracheal bifurcation. Although it could easily be seen through the bronchoscope, it could not be removed—the forceps he had all being too large to pass down so small a tube, but it was ultimately successfully removed with a hook which he specially designed for the purpose. He certainly thought blunt hooks ought to be used in selected cases.

Dr. PATERSON (in reply) said that the discussion had brought out what he wanted to know, and he was glad that his own opinion had been endorsed. He had been pleased to hear Mr. Howarth's remarks with regard to external operation. He (Dr. Paterson) had only seen the operation by an external route carried out once, and that was for a foreign body which had been impacted for from fifteen to sixteen years in the lower part of the gullet, just behind the heart. The question was, whether some thing ought to be done at once to save the patient's life, and a colleague of his under.

took the operation; and it was found—what one had suspected from the examination—that there was such dense adhesions that the safest policy was to leave the foreign body alone. In this case he was impressed by the readiness with which the foreign body could be approached at such a low level.

### **Diseases of the Thyroid Gland in their relation to Laryngology.**

By F. HOLT DIGGLE, F.R.C.S.

(ABSTRACT.)

IT is my intention to limit my remarks to the consideration of (1): the incidence of laryngeal paralysis in benign diseases of the thyroid gland and (2) the effects of thyroid enlargement on the position and shape of the trachea.

Various estimates as to the incidence of laryngeal paralysis in thyroid diseases have been quoted but it is difficult to ascertain what percentage of these is pre-operative.

During the last eighteen months I have examined thirty-four cases of thyroid enlargement, comprising twenty cases of simple parenchymatous goitre, seven adeno-parenchymatous, six exophthalmic and two cystic goitres. Of these thirty-four cases seven, or 20·6 per cent., presented laryngeal or tracheal manifestations. Probably this percentage is too high and a more reliable estimate would have been formed if a further series of cases had been examined. Of the seven cases presenting laryngeal and tracheal manifestations one was a case of functional aphonia occurring in a case of exophthalmic goitre. Three cases presented symptoms due to tracheal stenosis—one of which improved under medicinal treatment. There was one case of paralysis of the right vocal cord due to a perithyroiditis and one of adductor spasm due to a large parenchymatous goitre. Finally, one case with hoarseness was lost sight of before the investigation was completed. There was no laryngeal paralysis.

A study of the anatomical relations of the cervical portion of the recurrent laryngeal nerve will show that it is applied to the lateral wall of the trachea and does not lie in the tracheo-oesophageal groove as is so commonly believed. It will further be realized that a degree of pressure sufficient to implicate the recurrent nerves would compress the trachea and thereby induce symptoms requiring immediate attention before any severe paralysis could result.

Any extension of the thyroid gland behind the trachea, pharynx or oesophagus will tend to stretch the recurrent laryngeal nerve and so induce paralysis. An interesting case of this nature is recorded by James Berry. The patient was first seen by Sir StClair Thomson and presented a paralysis of one cord with some dysphagia. A large retropharyngeal extension of the thyroid gland was disclosed at the operation. These retrooesophageal, retropharyngeal or retrotracheal extensions of the thyroid gland are interesting, for if associated with laryngeal paralysis and some dysphagia, as is so commonly the case, they may be mistaken for malignant disease of the oesophagus or mediastinum without further and more thorough examination. The fact that in the majority of such extensions there is little or no apparent external swelling of the thyroid gland renders this mistake more easily made. Kaufmann quotes such a case where an oesophagostomy was performed, unfortunately with a fatal result.

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The recurrent laryngeal nerve may occasionally be involved in inflammatory adhesions.

In 1920 I saw a lady, about 60 years of age, who complained of pain and swelling of the right side of the neck with some hoarseness, of three days' duration. There was a hard tender swelling over the right lobe of the thyroid gland with paralysis of the right vocal cord. The hardness of the swelling and age of the patient at first suggested malignant disease but the short history and tenderness led me to regard the case as one of sudden hæmorrhage into a cystic goitre. She was kept under observation. The swelling and tenderness gradually subsided, and, when seen a few months later, the paralysis of the vocal cord had completely disappeared.

It will be seen, therefore, that in the diagnosis of malignancy too much reliance must not be placed on the fixation of a vocal cord.

By far the commonest complication of thyroid enlargements, however, is some displacement or torsion of the trachea. It is, as one would expect, far commoner than laryngeal nerve paralysis. In the majority of cases it is the young adolescent with the parenchymatous type of goitre who is so liable to respiratory distress from tracheal compression. The softer and more yielding nature of the tracheal wall in the young subject, together with the rapid increase in size of the parenchymatous goitre at the age of puberty, accounts for this complication. It is noteworthy that in the majority of such cases suffering from tracheal compression there is frequently only slight external swelling of the thyroid gland visible, so slight that occasionally it may be overlooked. Further, the patients are frequently unaware of any increase in size of the neck.

Substernal or intrathoracic goitres account for cases of laryngeal paralysis or respiratory embarrassment in older patients. It is noteworthy that in the recorded cases the left vocal cord has suffered more frequently than the right. This is as one would expect, having regard to the anatomy of the left recurrent laryngeal nerve. These intrathoracic goitres may be extensions from the normally situated thyroid gland or develop quite apart from the parent gland. They are interesting in that if associated with laryngeal paralysis and some distension of the superficial veins of the thorax, as occasionally is the case, they may, without further investigation, be set aside as inoperable cases of mediastinal tumours. The retrosternal dullness on percussion and diminished air entry lend support to this mistake.

[Various radiographs were shown illustrating the value of the X-rays in the diagnosis of tracheal compression and distortion with relief of the stenosis after partial thyroidectomy. It was pointed out that in the treatment of the bilaterally compressed trachea it was essential to remove much more thyroid tissue than would at first be thought necessary, otherwise one type of tracheal deformity would be replaced by another.]

### DISCUSSION.

MR. HERBERT TILLEY said he wished to relate two cases of great interest. One was that of a woman with left vocal cord paresis, which he (Mr. Tilley) thought was due to a thyroid tumour behind the sternum. Breathing was difficult, and the patient was hoarse. The difficult breathing was caused by pressure on the trachea, and the hoarseness by paresis of the left abductor. He (the speaker) sent the patient for operation to Mr. James Berry, who removed from underneath the sternum a very large

dermoid cyst. In this case the left vocal cord remained paralysed. He (Mr. Tilley) did not know what was the experience of members as regards this class of case, but he thought that when once recurrent paralysis of the vocal cord was definitely established, one very rarely found recovery from the paralysis after operation. The second case had occurred within the last three weeks. On examination of the larynx the glottis could scarcely be seen at all. On operation by Mr. Trotter a simple tumour of thyroid tissue was found underneath the thyroid cartilage and invading the ventricle. It was necessary to remove the greater part of the thyroid cartilage in order to remove the tumour, and the patient made a good recovery, though great anxiety was caused for the first forty-eight hours by severe intralaryngeal swelling. It was the only case of this kind he had ever known in which such an extension had invaded the larynx.

Mr. H. SMURTHWAITE said that during the last few years he had had the opportunity of seeing a large number of thyroid cases, and had examined each larynx to see if there was any paralysis before and after operation. He had not come across a genuine paralysis of the cords from simple thyroid enlargement.

Mr. DIGGLE (in reply) said that with regard to Mr. Tilley's remark about recovery after paralysis of the vocal cord, he (the speaker) was of the opinion that if the paralysis had existed longer than nine months there was little chance of recovery. With regard to Mr. Tilley's last remark about the removal of the thyroid, he thought that it had been shown that it was not sufficient merely to remove the isthmus of the thyroid.

### **Evolution of the Nasal Cavities and Sinuses in Relation to Function.**

By J. F. O'MALLEY, F.R.C.S.

(ABSTRACT.)

BEFORE the development of modern rhinology, anatomists and physiologists seemed to regard the structure and function of the nose as being exclusively concerned with the sense of smell. In man, the olfactory anatomy and its associated sense have retrogressed, whilst that of respiration and speech resonance, have assumed considerable importance. In tracing the comparative anatomy of the olfactory organ in the vertebrate group from the lamprey, through fishes, amphibia, reptiles, birds and lower mammals to man, many interesting phases of progression and retrogression in relation to function are noted.

First, an organ of smell only, then related to taste and assimilation, later to respiration, and as this predominates the more primitive function of olfaction tends to recede.

The specialized structure of the adult of a lower class becomes vestigial in the adult of a higher, but in the developing embryo of the higher it is always reproduced. Before the formation of the permanent palate establishes the nose to the mammalian type, the human foetus goes through phases we find in the lamprey, dog-fish, amphibia, reptiles and birds. As Keith puts it, there are three stages: (1) A piscine, in which nose and mouth are formed independently; (2) an amphibious stage, where the nasal respiratory passage opens on the roof of the mouth; and (3) a mammalian stage, in which it opens into the nasopharynx.

The evolutionary changes in the nose centre around variations in the following structures: (1) Olfactory sac and brain connections; (2) epithelium



—olfactory, respiratory; (3) Jacobson's organ; (4) external nares; (5) internal nares (or choanæ); (6) nasal cavities; (7) nasal septum; (8) nasal floor; (9) nasopalatine foramina; (10) turbinates (or conchæ); (11) erectile tissue; (12) intranarial epiglottis; (13) nasal accessory sinuses.

The first instance of paired nasal cavities, with external and internal openings, joined by short passages, is seen in lung fishes, all other fishes having external openings only, which lead to pits containing sacs of sensory epithelium. The short paired nasal passages of the lung fishes become the nasal chambers or cavities in amphibia, reptiles, birds and mammals. In amphibia, birds and mammals they are arranged in a dorsal or olfactory and ventral or respiratory compartment and in reptiles in an inner or outer compartment with similar functions. The single outer opening of the lamprey leads to a large sac divided by a septum; this is probably the forerunner of the septum in all paired-cavity noses. The extent of sensory epithelium has a direct relation to the power of smell, though the size of the brain connexions concerned with olfaction, are of greater importance. In the human foetus at the fourth month, the sensory epithelium occupies, relatively to the size of the nasal chamber, the same proportion of surface as that seen in the adult chimpanzee and other animals with a powerful sense of smell.

Jacobson's organ, first seen in amphibia and attaining its highest development in herbivora, but vestigial in birds and man, is a specialized portion of the olfactory epithelium, thrust to the lower part of the septum near the nasopalatine openings, where it can receive odours of juices from the mouth.

Turbinates or conchæ, the first trace of which is seen in amphibians of the urodele order, become two in number in reptiles, three in birds, man, and most mammals, but six in the chimpanzee and animals with keen sense of smell, and in the human foetus up to the fourth month.

Erectile tissue found in all warm-blooded animals, but most extensive in its development in man, is closely related to the beginning of sexual life and the advent of adolescence, though not sexual in function, but respiratory. Wright attributes this excessive development in man to his clothing, the warmth of his dwellings and general artificial environment.

The intranarial epiglottis is normal in the adult whale and in the human foetus up to the fifth month. In many animals the epiglottis is in the nasopharynx in contact with the palate, but in man it is withdrawn to make provision for the function of speech. A large tongue with epiglottis near the palate in man causes muffled speech.

The nasal accessory sinuses are the outcome of the need for bulk without addition to weight, on the hollow girder principle, to widen the base of the skull. They are concerned with the development of the permanent teeth, respiration and speech resonance. The growth backward of the antrum of Highmore brings down the permanent double teeth; the mucous lining covered with ciliated epithelium completes the watery saturation of the warm air entering the cavities and the bulging forward of the cranial roof and downward inclination of the face, brings the sinuses in front of the sound-producing mechanism in the larynx to get the maximum resonating effect.



## A Clinical Note on the After-treatment of Empyema of the Maxillary Antrum (Denker's Operation).

By D. LINDLEY SEWELL, M.B.

(ABSTRACT.)

Mr. SEWELL said that he believed Denker's operation to be the most efficient because of the facilities which it offered for the complete inspection of the antral cavity. He did not pack the cavity after completion of the operation, but only the mucous membrane flap from the antro-nasal wall, which was held down in position by a strip of ribbon gauze. This was removed on the fourth day, and in the majority of cases no further packing was employed, nor was the antral cavity irrigated. Mr. Sewell regarded irrigation as impeding rather than helping the recovery of the lining mucous membrane of the antrum, and considered that it should be employed only in the minority of cases—those in which recovery was slow. He had found solutions of silver nitrate very useful in the treatment of indolent cases.

### DISCUSSION.

Sir WILLIAM MILLIGAN said that it was his custom to perform the Denker's operation. The lines of treatment had been sketched out very well by Mr. Sewell. There were two points before the Section, viz.: (1) Whether irrigation treatment should be carried out after operation, or not; (2) the method of dealing with the flap. The only question he would like to ask Mr. Sewell was in connexion with cases in which a concomitant ethmoidal affection was present which frequently kept up a certain amount of intranasal discharge. He (the speaker) did not allow the antrum to be irrigated for ten days. He thought it essential that the buccal opening should be allowed to heal at once. He never packed the antrum.

Mr. BANKS-DAVIS said they were all indebted to Mr. Sewell for his interesting and practical paper. He was interested in his method of non-washing out, as he (the speaker) adopted that method himself. Had Mr. Sewell any special means of dealing with the crusts which formed in the nose after operation and were often so distressing to the patient?

Mr. T. B. LAYTON said that since he paid a visit a few years ago to see Sir William Milligan operate, he had never sewn up the buccal wound, and his patients seemed to have far less swelling of the face; the wound also seemed to heal more quickly. He was much interested to learn that Mr. Sewell opened up the wound four days later, and removed the packing. He had always taught his patients to wash out the maxillary antrum after operation. For that reason he removed a piece of the inferior turbinal, because in a case in which he had not done so, he had found it difficult to teach the patient to pass the cannula afterwards. He had come to the conclusion that the question of washing was not so important as that of the solution used, and that physiological saline was a better lotion for the nose than any antiseptic lotion. He doubted if it were possible to put into the nose any antiseptic which had the slightest effect on the micro-organisms, unless it was so strong that it did harm to the mucous membranes. He, personally, found hypotonic solutions irritating to the nose. Hypertonic solutions were less so, but he believed that physiological saline was better than either.

Mr. J. F. O'MALLEY said he always made an opening under the anterior end of the turbinates much in the same manner as had been described. He never made a buccal opening, and never washed out the antrum. The only point in favour of washing-out was that it had a very satisfying effect upon the patient.

Mr. A. J. M. WRIGHT said he thought that the wound healed up much better without a buccal opening. With regard to washing out, he had never been able to

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discontinue it entirely. He still found there were cases of chronic antral suppuration where one had to carry out post-operative washing out.

Dr. DOUGLAS GUTHRIE said that in a great many cases so severe a procedure as a Denker's operation was not required, provided a sufficient amount of the anterior wall of the canine fossa was removed in the more usual Caldwell-Luc operation. In the Canfield technique the operation was practically Denker's performed intranasally. He suggested the use of an oily spray in the after-treatment to prevent the formation of crusts.

Mr. GRAHAM BROWN said he thought there was more sinus trouble in Queensland than in this country. In the previous year he had performed sixteen antral operations through a buccal opening. In a certain number of these antral operations the result was not satisfactory; there was some persistence of muco-purulent discharge. He (the speaker) thought the thing to aim at first was a good view of the antral cavity; and for this reason during the last five years he had been doing all his antral operations under local anaesthesia. By this method there was practically no bleeding. He did not remove any portion of the inferior turbinal. He never did a Denker's operation. He did not think a flap was essential; he had tried it, and did not think much was gained by it. He packed the antrum with a rubber glove, which was removed within twelve hours; he began washing out on the fourth day, when he invariably found that the buccal wound had healed. For washing out, he used eusol solution (1 in 6), and followed that almost immediately with a lotion practically identical with Wright's solution. He persisted with the washing out, gradually decreasing the number of applications. He thought it was an essential procedure; it was a mistake to leave an antrum unirrigated. A point to consider was that frequently the anterior ethmoidal cells were involved in the suppurative process, and these required attention.

Dr. IRWIN MOORE said he never carried out any after-operation packing, nor did he suture the buccal incision. In most of his cases he operated by the intranasal route, which was in his experience satisfactory in the majority of cases; the chief point to aim at to ensure success was to make the nasal opening as large as possible. He avoided washing out for four or five days, and then he invariably used an ordinary saline solution, and later, he found the best treatment was to wipe out the antrum occasionally with argyrol (25 per cent.).

Mr. SEWELL (in reply) said he did not make a practice of suturing the buccal wound, because he had never found any difficulty in the natural closing of the wound except in two cases. In the usual antral case he had never had any trouble about the closing of the wound. As regarded the formation of crusts, these were very difficult to deal with, and what he found most efficacious was to leave in over night a piece of gauze with vaseline as carried out in the treatment of atrophic rhinitis. In certain cases where there was not only suppuration of the antrum, but extensive ethmoidal disease and frontal sinus as well, the great trouble was crusting. He thought Mr. Guthrie had really described a Canfield's operation. He (Mr. Sewell) had only tried it on two occasions, and it seemed to him that that operation was merely a Denker's carried out through the nose—that is to say, under more difficult conditions.

### Some further Remarks on the Reduction or Destruction of Hypertrophied or Diseased Tonsils by means of Caustic Soda and Slaked Lime (London Paste).<sup>1</sup>

By IRWIN MOORE, M.B.

(Supplementary to a Paper read at the First Annual Summer Congress of the Section of Laryngology, May 2, 1922, and published *in extenso* in the *Journal of Laryngology and Otology*, 1919, xxxiv, p. 387.

<sup>1</sup> Owing to lack of time this paper was not read. It will be published later in the *British Medical Journal*.

**Demonstration illustrating certain Pathological and Surgical Points in the Treatment of Malignant Disease in the Upper Jaw.**

By E. MUSGRAVE WOODMAN, F.R.C.S.

SLIDES were exhibited showing the microscopic appearances, under both low and high powers, of the various types of malignant tumour found in this region. Attention was called to a particular type of tumour of a general acinous formation in which characteristic cells were conspicuous by their vacuolation. These tumours had been diagnosed by Professor Shattock as malignant endothelioma. Sections of a cross-section of a normal ethmoidal labyrinth were demonstrated and the remarkable resemblance to the tumours mentioned above was pointed out. Two series of coloured photographs of operations were recorded as illustrating the extensive growths which can now be dealt with successfully. The first depicted an extensive epitheliomatous ulcer below the eye, which necessitated the removal of the eyeball and exposed the dura mater of the base of the brain; the second showed an extensive growth of the antrum, which had perforated the skin of the face below and above the orbit. Photographs taken both before and after operation were exhibited.

**DISCUSSION.**

Mr. W. S. SYME said that he had several times operated on malignant growths of the upper jaw. In one case of sarcoma (upper jaw, sinuses and nose) in which he thought he would get the best view, he made the incision down the middle of the face and split the nose. In that way he was able to get into all the accessory cavities and remove the growth from the sinuses. Very little disfigurement occurred, and the patient lived many years afterwards. He (Mr. Syme) considered that in dealing with these cases one owed a great deal of the success to diathermy. He was not convinced as to the value of radium in these cases. He had seen great destruction caused by radium, and, though he did not know what the experience of others had been, he, personally, would be very chary in using radium.

Mr. G. WILKINSON asked Mr. Woodman whether, in the case of rodent ulcers which had become epitheliomatous, he found it necessary to dissect the glands in the neck, or if it was usual for the glands to escape infection in this class of case.

Mr. MUSGRAVE WOODMAN (in reply) said that he considered this work came well within the specialty, because general surgeons without intimate knowledge of the anatomy of these air cells were not in a position to eradicate malignant disease of the upper jaw. He agreed that the effect of radium was very destructive. One of his patients had a very extensive sarcoma of the jaw which was removed, and was followed by recurrence in the cheek. He (Mr. Woodman) applied 150 mg. of radium, and a month later the whole of the front of the face fell out. In reply to Mr. Wilkinson, he was of opinion that glands of the neck did not tend to become involved, but in all cases of epithelioma, whether the glands were obviously affected or not, they should be excised widely and completely on the same side of the neck.

**Malignant Disease of the Nasopharynx, with special Reference to its Malignancy and to its Treatment—Operative and Non-operative.<sup>1</sup>**

By Sir WILLIAM MILLIGAN, M.D.

<sup>1</sup> Owing to lack of time this paper was not read. It will be read before the Section during the coming Session.

### **Sarcoma of the Left Tonsil.**

By Sir WILLIAM MILLIGAN, M.D.

R. H., MALE, aged 57. Difficulty in swallowing for six months. Loss of weight, anaemia. Deafness left ear. Firm lobulated growth springing from the left tonsillar region, filling mouth and displacing soft palate. Enlarged cervical gland freely movable.

May 2, 1923: Microscopic report of a portion removed: Round celled sarcoma.

May 12, 1923: Insertion of radium tube 40 mg.

May 26, 1923: Almost complete disappearance of growth. General condition improving.

Sir WILLIAM MILLIGAN (in reply to questions) said he had not done anything more than apply radium in order to demonstrate its very rapid and immediate effect. He intended to remove the rest of the tonsil by diathermy.

### **Epithelioma of Soft Palate and Left Anterior Faucial Pillar.**

By Sir WILLIAM MILLIGAN, M.D.

H. H. M., MALE, aged 59. Sore throat and otalgia of three months' duration. Ulcerated and vascular growth involving portion of the soft palate, left anterior faucial pillar and posterior surface of the uvula. Infiltration of tissues; no palpable glands.

Microscopic examination: Epithelioma.

May 25, 1920: Diathermy under general anaesthesia.

June 2, 1923: Complete freedom from all symptoms since June, 1920. Small nodule now seen upon the posterior pharyngeal wall, with a hard and indurated base. No pain.

#### **DISCUSSION.**

Mr. W. S. SYME said that the whole question of diathermy was so extensive that one hesitated to say much. In this case there certainly appeared to be a recurrence which one would be well advised to remove by diathermy. One difficulty he (the speaker) had found in connexion with these cases was that the scar formed was very firm, and that the jaws became firmly fixed together. He would like to know if others had had the same experience. In one of his cases operated upon by diathermy a year ago there was a growth declared inoperable by ordinary methods—involving both upper and lower jaws, also a large mass of glands. No recurrence followed in the mouth. Several months later the glands atrophied to the size of the thumb or smaller and became very hard and firm. They were removed and found to be fibrous. He wondered if others had seen these changes—which were evidently a process towards cure—taking place in the glands after removal of the primary growth by diathermy.

Sir WILLIAM MILLIGAN (in reply) said that there was a small recurrence on the posterior wall which he intended to diathermize at once. He had observed fixation of the jaw after extensive diathermy and did not know what to do in such cases except to advise exercise. With regard to the question of removal of the glands, when glands were enlarged, he usually dissected them out by a block dissection.

**Epithelioma of the Right Vocal Cord: Laryngofissure.**

By Sir WILLIAM MILLIGAN, M.D.

T. J., MALE, aged 62. First seen June 14, 1915, complaining of loss of voice for twelve months. Examination: Cauliflower growth occupying posterior two-thirds of the right vocal cord, which was fixed.

June 20, 1915: Laryngofissure; cord removed. Alar cartilage not removed.

Microscopical report: Epithelioma.

June 6, 1923: No symptoms beyond a husky voice.

The Section were of opinion that the result in this case was excellent.

**Laryngeal Growth.**

By Sir WILLIAM MILLIGAN, M.D.

W. A. D., MALE, aged 56. Progressive loss of voice for nine months, and slight expectoration. No bacilli found. History of left-sided pleurisy and gradual loss of weight—(radiogram of chest shown)—enlarged gland in neck. Opinions requested as to exact diagnosis and as to treatment.

**DISCUSSION.**

Mr. HERBERT TILLEY thought the case was one of chronic tuberculosis, first, from the clinical appearances of the larynx, and secondly, because the patient had a rapid pulse, and looked a "sick man." He suggested that the evening temperature be taken for a week.

Mr. A. J. M. WRIGHT said he was also of the opinion that this was a case of tuberculosis. It reminded him of a male patient, a somewhat older man than the case shown, with no definite signs of tuberculosis in the chest. He (the speaker) had performed a laryngo-fissure on the assumption that the case was malignant. The patient very rapidly went down hill and died shortly afterwards of pulmonary tuberculosis.

Mr. H. J. BANKS-DAVIS said that the patient was referred to him as a case of laryngeal paralysis, considered by his physician as thoracic in origin. When he (Mr. Banks-Davis) first saw the patient there was some swelling of the left ventricular band in addition to the laryngeal paralysis. When he next saw him there was no question as to the swelling. He again saw him a month ago and the œdema had increased. At first he thought it might be a case of malignant disease of the larynx, but now considered it was tuberculosis. Whether there was any malignant disease in conjunction with it, it was impossible to tell. He would be very chary of doing anything to the larynx unless a portion of the swelling was examined microscopically first.

Mr. W. S. SYME said he considered that the lesion was more extensive than one would expect to find with tubercle; the colour and appearance generally being more suggestive of malignancy than of tubercle. He advised that nothing should be done until a definite diagnosis had been made.

Sir WILLIAM MILLIGAN said he was of the opinion that it was a mixed infection—a chronic tuberculosis which had become malignant. No tubercle bacilli had been found in the sputum. There were no clinical signs of tuberculosis of the lungs, and the patient had a sub-normal temperature. Again, there was a certain amount

of fixation on that side, as noticed by Mr. Syme—an amount not usual in a case of tuberculosis, and that suggested superadded malignancy. There was also very rapid loss of weight—much more than one would expect to see in tuberculosis. He thought there was also some superficial ulceration. He certainly did not propose to do a laryngo-fissure, but to remove the gland in the neck first and have it microscoped. If it proved to be malignant, then he would remove a small portion of the growth by the direct method, have it microscoped, and be guided by the result. He thought it might prove to be a mixed infection.

*Postscript.*—A portion of tissue was removed and microscopically showed tuberculous disease without any evidence of superimposed malignancy.

### **Suppurative Disease of Left Frontal Sinus and Left Maxillary Antrum.**

By Sir WILLIAM MILLIGAN, M.D., and D. LINDLEY SEWELL, M.B.

K. F., MALE, aged 29. Bilateral nasal purulent discharge for four years. Left-sided frontal headache. Polypi and pus in left middle meatus.

X-ray report: Left frontal and maxillary sinuses opaque.

Operation, June 17, 1922: Denker's operation on left maxillary antrum, and left frontal sinus obliterated.

### **Tuberculous Growth in Left Naris.**

By Sir WILLIAM MILLIGAN, M.D., and D. LINDLEY SEWELL, M.B.

M. M., FEMALE, aged 32. Gradual blocking of left nasal passage with widening of nasal bridge.

Examination: Papillomatous looking mass springing from region of vestibule and anterior portion of nasal septum. Septal spur removed from left nasal passage three years previously. Is traumatic infection a feasible proposition?

Growth removed and base seared with galvano-cautery point.

Microscopic examination: Tubercular growth with caseation.

### **Orbital Cellulitis: Invasion of Frontal Sinus; Osteo-myelitis of Frontal Bone.**

By Sir WILLIAM MILLIGAN, M.D., and FRANK WRIGLEY, M.D.

W. M., MALE, aged 15. First seen, April 18, 1923. Two days' illness. Left eye proptosed. Conjunctiva injected and chemotic.

April 19, 1923: Left frontal sinus considered normal.

April 20: Incision through upper eyelid, pus evacuated.

April 21: Marked œdema over frontal region.

April 22: Examination of pus shows a mixed infection, streptococcus

and staphylococcus. Left eye eviscerated. Left frontal sinus opened—no pus found.

April 28: Condition of patient serious. Œdema spreading over forehead, three incisions made over scalp down to bone.

May 1: Temperature high, rigidity of neck, Kernig's sign, right side.

May 4: High temperature, rapid pulse, œdema over whole of left side of frontal region. Copious discharge of pus from orbit.

May 5: Operation (Sir William Milligan). Frontal sinus reopened and found full of pus and granulation tissue. Outer table of skull removed over left frontal area—whole of wound painted with pure carbolic acid and left open. Fomentations applied.

Gradual recovery from time of operation to date, the only drawback being the development of an abscess in the neck which has now been drained.

#### DISCUSSION.

Mr. J. F. O'MALLEY said the case reminded him of a patient whom he had seen some years ago in a fever hospital. A diagnosis had already been made of frontal sinus disease but on examination he (the speaker) had found pus in the ethmoidal region, and had come to the conclusion that the trouble had started in that region and was due to the effect of changes in the mucous membrane induced by the scarlet fever.

Sir WILLIAM MILLIGAN (in reply) said that he understood from Mr. Wrigley that there was no accessory sinus disease at all. He (Sir William) did not know the cause of the orbital cellulitis. The boy was very ill when he was sent to the infirmary. He (the speaker) opened the frontal sinus which he found infected, removed the outer table of the skull, and applied pure carbolic acid to the raw surfaces. The boy had made good progress, and he hoped would recover altogether. Examination of the pus showed a streptococcal infection.

*Postscript.*—July 23, 1923. The boy is now quite well.

### **Sarcoma of Right Tonsil and Surrounding Faucial Region.**

By Sir WILLIAM MILLIGAN, M.D., and FRANK WRIGLEY, M.D.

A. J., MALE, aged 34. Swelling in throat accompanied by slight attacks of spontaneous hæmorrhage. Ragged and ulcerated hyperæmic mass occupying right side of fauces; firm, fixed, involving tonsil, pillars of fauces and adjacent area of tongue. Mass spreads half way over oro-pharynx, and for some distance down the pharynx. No palpable glands.

Microscopic report of portion removed. Large round-celled sarcoma. Tracheotomy-diathermy. December 22, 1921: Tracheotomy tube removed.

December 25, 1921: Referred to Radium Institute for external application of plates.

### **Two Cases of Chronic Œdema of Orbit.**

By F. H. WESTMACOTT, C.B.E., F.R.C.S.

(1) R. W. B., MALE, aged 53. Odema of left eyelid since January, 1921. Condition stationary since March, 1921. No pain, no nasal discharge, accessory sinuses clear. Fundi normal. Diagnosis invited



## 92 Westmacott: *Edema of Orbit; Sarcoma of Frontal Bones*

(2) S. A., female, aged 42. Edema of right eyelids commenced five years ago at inner canthus. No pain. Sight not affected. Suppuration from right middle meatus. November, 1921: Ethmoidal and sphenoidal sinuses opened. Pus present. No opening found into the orbit from ethmoidal labyrinth. Swelling varies but is less than in 1921. Wassermann negative. Diagnosis invited.

### DISCUSSION.

Mr. HERBERT TILLEY referred to a similar case which he had shown to the Section in London some five or six years previously, of a male who had served in the South African War, and had had malaria several times. In this case the same condition was present, except that it was bilateral, and he concluded that there was some local affection of the lymphatics. He suggested a bacteriological examination of the lymphatic fluid from the oedematous area.

Mr. A. J. M. WRIGHT said he had seen a similar condition in a girl aged 14. She had a marked degree of conjunctivitis, and it seemed to him (the speaker) that this was a possible means of entry of the infection.

Sir WILLIAM MILLIGAN said that he also regarded the condition as a chronic lymphangitis. There was a history of injury. He (the speaker) had seen two very similar cases, in both of which there had been a history of injury—abrasion and infection. In both those cases tissue had been removed and microscopically examined, and was found to be chronic lymphangitis. He considered that both of the cases now shown were of a similar nature, and suggested microscopical examination of a piece of the tissue.

Mr. WESTMACOTT (in reply) said he had hoped to elicit more information. In the first case, the man had not only had a mosquito bite over twenty-three years ago, but was also knocked in the eye by a cricket ball, and it was quite probable, as Sir William Milligan had suggested, that there had been some chronic endo-lymphangitis. The nasal sinuses in this case were free. With regard to treatment, he was very grateful for the suggestions made. He would have the fluid examined, and also a piece of the tissue.

## Sarcoma of Maxilla and Malar and Frontal Bones.

By F. H. WESTMACOTT, C.B.E., F.R.C.S.

W. P., MALE, aged 37. Sarcoma of maxillary antrum involving orbital plate, canine fossa, malar bone and external angle of frontal bone. Excised on September 13, 1913, together with the ethmoidal labyrinth and opening up of sphenoidal cavity. Microscopic section showed round-celled sarcoma. No further symptoms until February 23, when the cheek bone became hardened and the eye proptosed, and there was pain in the first division of the fifth nerve. After application of radium the pain subsided and some prominence of the part receded.

### DISCUSSION.

Mr. E. MUSGRAVE WOODMAN said this was a remarkable case, in which a very successful operation had been performed ten years ago, since which the patient had remained apparently perfectly cured. Then, possibly from a neglected ethmoidal cell or from a small focus in the malar bone, recurrence occurred across the orbital bone and appeared below the eyelid. This was quite unlike round-celled sarcoma. It did not lie low for ten years and then slowly creep up. He thought Mr. Westmacott at the present time need be in no anxiety about his patient. The eyelid was free from growth, and he advised excision of the eyeball and elimination of the growth. In a

case of this kind, in which the palate was not involved, he suggested leaving a window in the palate through which the involved area could be from time to time inspected, or through which radium could be inserted at any period after the operation. The opening could be easily covered up by a dental plate.

Mr. WESTMACOTT (in reply) said he was glad to have Mr. Woodman's advice about this case, and would, in future operations, cut out a window in the palate.

### **Extensive Osteomyelitis of Frontal Bone.**

By F. H. WESTMACOTT, C.B.E., F.R.C.S.

T. J., MALE, aged 40. March, 1922: Pain in right frontal sinus with purulent nasal discharge for several weeks. Turbinal resection—opening of ethmoidal labyrinth internally on April 29, 1922. October, 1922: Fluctuant swelling over whole frontal area.

Operation, October 4, 1922: Transverse frontal incision. Extensive cario-necrosis outer and inner tables of frontal bone with dura mater exposed the size of a shilling. Both frontal sinuses opened to orbital margin and below it. Tube inserted in left sinus and division removed. Recovery.

Polypoid mass now appearing in ethmoidal area. No discomfort.

### **Tuberculosis of the Larynx.**

By J. A. KNOWLES RENSCHAW, M.D.

J. W., MALE, aged 55. Extensive pulmonary tuberculosis for seven years. Secondary laryngeal tuberculosis appeared in 1919. Both vocal cords were affected throughout their length. There was much hyperplasia and congestion. Some ulceration took place over the left vocal process.

Treated by vocal rest, intratracheal injections of liq. iodi. (M. and J.) and formalin sprays. The vocal cords resumed a normal outline. Slight congestion on the right side remains. The tuberculosis of the lung has been active throughout the treatment and still persists.

Mr. J. ARNOLD-JONES, F.R.C.S., exhibited the following cases and specimens:—

#### **CASES.**

(I) Extensive Tuberculosis of the Nose, Pharynx and Larynx. Treated by Local Measures and Tuberculin.

(II) Nasopharyngeal Sarcoma. Surgical Removal ten years ago. After-treatment with Radium; no Recurrence.

(III) A case of Benign Growth in the Posterior Commissure of the Larynx.

#### **SPECIMENS.**

(A) Microscopical Sections of a Hæmangioma removed from the Left Vocal Cord of a Male, aged 45, by the Indirect Method.

(B) X-ray Photographs illustrating Involvement of the Left Recurrent Laryngeal Nerve by a Mediastinal New Growth producing Paralysis of the Left Vocal Cord.

(C) X-ray Photographs showing a Spinal Abscess which produced intense Stridor by Compression of the Trachea.

**Double Abductor Paralysis.**

By A. A. SMALLEY, M.B.

C. S., A MALE, aged 50. In February last difficulty in breathing and hoarseness began and continued for two months. Inspiratory stridor, cyanosis. Tracheotomy considered. Almost complete abductor paralysis; cords congested and rough. Scars on soft palate. Wassermann positive, 1/40. No evidence of other central or peripheral nervous lesion. Improved under treatment, but still injection of cords and abductor par.

Mr. HERBERT TILLEY advised extraction of teeth.

**Endothelioma of Ethmoid: Lateral Rhinotomy.**

By A. A. SMALLEY, M.B.

J. H., AGED 54. Operation, November, 1920. Ligature of external carotid. Lateral rhinotomy. Removal of tumour, antro-nasal wall, and lateral mass of ethmoid. Antrum, sphenoidal and frontal sinuses were not involved. Patient, tumour and section shown.

**Carcinoma of Antrum: Removal of Upper Jaw.**

By A. A. SMALLEY, M.B.

M. C., AGED 67. September, 1920: Swelling of left cheek for two months. No pus or growth seen in nose, but bulging of antronasal wall. Antrum opaque to X-rays. Slightly enlarged gland in each anterior triangle. Operation: Ligature of external carotid artery, removal of inflamed gland, removal of superior maxilla, including orbital floor, also lateral mass of ethmoid. Growth had invaded skin of cheek, which had to be very thinly undercut.

**Nasopharyngeal Tumour: ? A Cystic Adenoma, containing Cartilage (Wet Specimen and Section).**

By A. A. SMALLEY, M.B.

REMOVED *per vias naturales* from a woman aged 46. Very small attachment to posterior border of septum high up. No sign of recurrence after two years.

**Chronic Cellulitis of the Face.**

By G. E. ARCHER, M.B.

J. T. S., AGED 26. Intranasal operation, 1920 (? attempted submucous resection), followed by œdema of the face. First seen by exhibitor, September, 1922. Chronic cellulitis of the face, with periodic exacerbation.

Oedema never completely disappeared. Perforation of the septum, adhesions between right inferior turbinal and septum: no accessory sinus disease. Condition failed to yield to treatment, including autogenous vaccine.

December, 1922: Patch of alopecia area developed.

February, 1923: Intranasal adhesions severed. Facial massage and faradism instituted. Since then condition has slowly but distinctly improved.

### **Case of Branchial Cyst.**

By F. HOLT DIGGLE, F.R.C.S.

FEMALE, aged 40. Had noticed a tender swelling under the lower part of right sterno-mastoid, for five weeks; getting bigger. On account of extreme hardness and fixation, thought to be malignant: there is a discharging cervical sinus in a similar position on the left side and there is a dimple over the swelling on the right side.

### **Case of ? Clinically Malignant Disease of the Left Pyriform Sinus.**

By F. HOLT DIGGLE, F.R.C.S.

MALE, aged 54. Early in 1922 noticed hoarseness and difficulty in swallowing. No improvement in spite of treatment from own doctor.

September 21, 1922: Epiglottidean fold infiltrated and oedematous; bulging of left wall of larynx preventing view of left cord. A mass of whitish granulations projecting from left pyriform sinus to outer side of above swelling. Wassermann negative.

Oedema, fatty degeneration of the heart and chronic Bright's disease prevented operation.

October 18, 1922: Three radium tubes, each 1.5 millicuries, inserted into left pyriform sinus (direct method, local anaesthesia).

January 23, 1923: Four tubes inserted, each 1.5 millicuries, under direct laryngoscopy. Since then two external applications of radium plates at intervals of six weeks.

May 17, 1923: Symptoms have disappeared. There is marked diminution of the swelling and a perfect view of the larynx is obtained. No limitation of movement of left vocal cord.

### **Epithelioma of Right Vocal Cord.**

By F. HOLT DIGGLE, F.R.C.S.

FEMALE, aged 58. Complained of hoarseness for eight months. Tumour involving anterior two-thirds right vocal cord, which moves slightly on phonation.

October 23, 1921: Laryngo-fissure. Thyroid ala retained. Right laryngeal artery ligatured.

Histological report (section shown): Epithelioma of vocal cord.

## DEMONSTRATIONS.

P. WATSON-WILLIAMS, M.D., A Method of Exploration of the Nasal Accessory Sinuses.

IRWIN MOORE, M.B., The Reduction or Destruction of Hypertrophied or Diseased Tonsils by means of Caustic Soda and Slaked Lime (London Paste).

J. M. W. MORISON, M.B., Cardiospasms and other Diseases of the Esophagus.

E. W. TWINING, M.R.C.S., L.R.C.P., Pirie's Method of Radiographing the Mastoid Cells.

C. C. ANDERSON, M.B., Deep X-Ray Therapy in Laryngological Conditions  
(At the X-ray Department of the Royal Infirmary.)

A. BURROWS, M.D., Methods of Application of Radium (cases shown).  
(At the Radium Institute.)

The following cases, with the Discussion, have been referred for later publication when further investigations or completed reports have been submitted:—

Sir WILLIAM MILLIGAN, M.D., "(?) Papilloma of Faucial Mucous Membrane and Enlarged Cervical Glands."

F. H. WESTMACOTT, C.B.E., F.R.C.S.: (1) "Growth in Anterior Commissure and on adjoining Extremity of Right Vocal Cord."

(2) "Growth in Anterior Commissure."

(3) "Laryngeal Growth."

(4) "Ulceration of Soft Palate and Left Tonsil."

(5) "Cicatricial Contraction of Right Vocal Cord."

(6) "Growth in Anterior Commissure."

(7) "Growth in Anterior Commissure of Larynx and on Right Vocal Cord."

(8) "Lupus of Right Nasal Fossa."

J. A. KNOWLES RENSHAW, M.D.: (1) "Difficulty in Enunciation and Dysphagia."

(2) "Swelling of Right Side of Palate."

F. G. WRIGLEY, M.D.: (1) "Malignant Disease (? Sarcoma), of Left Antrum."

(2) "Proptosis of Right Eye; with Post-nasal Catarrh, Adenoids and enlarged Tonsils."

(3) "Paralysis of Right Vocal Cord."

G. E. ARCHER, M.B.: (1) "Tertiary Syphilitic Lesions of Tonsils."

(2) "Laryngeal Polyp."

(3) "Lupus of Inferior Turbinals."

F. HOLT DIGGLE, F.R.C.S.: (1) "(?) Tuberculosis of Larynx."

(2) "Papilloma of Left Vocal Cord."

(3) "Laryngeal Papillomata."

R. LINDLEY SEWELL, M.B.: (1) "Epithelioma of Anterior Pillar of Fauces and Base of Tongue."

(2) "Abductor Paralysis of Left Vocal Cord."

(3) "Extensive Tuberculous Disease of Pharynx and Larynx."

(4) "Lupus of Anterior End of Inferior Turbinal."

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**VOLUME THE SIXTEENTH**

SESSION 1922-23

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SECTION OF MEDICINE



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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

## Section of Medicine.

President—Dr. G. NEWTON PITT, O.B.E.

### The Nervous and Mental Disorders of Severe Anæmias in Relation to their Infective Lesions and Blood Changes.

By WILLIAM HUNTER, C.B., M.D., F.R.C.P.

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#### SECTION I.—GENERAL CHARACTER OF CASES AND PROBLEMS.

##### (a) *Introductory.*

THE subject of this paper has been one of great interest and much perplexity as regards, *first*, the character of the nervous features presented in certain cases of anæmia; *secondly*, their relation to the anæmia, which they appear sometimes to precede, sometimes to accompany, sometimes to follow; *thirdly*, the character of the anæmia with which they are associated—whether the so-called “primary” or “idiopathic” type, commonly called “pernicious” (A); or simpler and various so-called “secondary” types (B, C, D, E, F). The difficulties regarding the latter have been much increased by the varying and similar characters of the blood changes which anæmias both primary and secondary may present in different cases, or in the same case at various times; variations which render it impossible at times to decide, by blood changes alone, to which type the case belongs (*see* Table I, p. 4).

## 2 Hunter: *Nervous and Mental Disorders of Severe Anæmias*

The anæmias with which nervous and mental disorders may be associated are the two definite types possessing the following identity:—

(A) *Pernicious anæmia* (seasonal, glossitic, hæmolytic, hyperplastic) (see Table II, p. 28).

(B) *Septic anæmia* (non-seasonal, non-glossitic, aplastic). The types of blood changes in the two forms are shown in Table I (p. 4), also in Table IV (p. 35).

The degree of nervous disorder that may be associated with these two types is illustrated by two cases recently seen.

*Type A.*—A gentleman seen about two years ago. "He has had his ups and downs, and has on at least two occasions been practically insane, with delusions of grandeur and suspicion. I had a mental specialist down to see him early this year, but we did not send him away, and eventually he calmed down a good deal."

*Type B.*—A gentleman, aged 70, seen November, 1921. General health good till 1914, when he had a "nervous breakdown," for which he was sent to a home for six months. He got well till Christmas, 1920, when he again got low spirited, and was sent at first to a home (six weeks), then to a mental hospital (two months). He lost some flesh, but showed no anæmia, and seemed perfectly well (July and August, 1921). Then in October he suddenly became increasingly pale and breathless, and was found on October 25 to have—

	Blood Report number	My clinical interpretation	
		Normal	Percentage
Red cells ... ..	2,250,000 ...	5,000,000 ...	50
Hæmoglobin ... ..	30 ...	100 ...	30
Colour index ... ..	0·6 ...	1·0 ...	0·6
Leucocytes ... ..	2,000 ...	7,500 ...	26
Polymorphs (51 per cent.) ... ..	1,020 ...	5,000 ...	20
Lymphocytes (37 per cent.) ... ..	740 ...	2,000 ...	37
Hyalines (12 per cent.) ... ..	240 ...	300 ...	80
Eosinophils ... ..	0 ...	200 ...	0

When seen by me on November 3, 1921, he was desperately ill and profoundly anæmic; he died about a fortnight later.

The questions involved in these two cases were: (1) What was the nature of their nervous features? (2) What were their relation to the anæmia which in the first case preceded and in the second case followed their original onset? (3) What was the nature of the anæmias associated with them?

The subject matters thus relate to points of very wide interest to very different groups of observers. This wide range renders it difficult for an observer in any one class to interpret his results in his own sphere of work; and still more difficult to interpret them in their relation to the work in other spheres. The neurological observer may find it difficult to interpret his findings in relation to other clinical ætiological or blood features of his cases. The ordinary clinical observer is not specially qualified to speak of the neurological aspects of the case; even in his own clinical observations and diagnosis he has too frequently to fall back upon information regarding blood changes supplied by the clinical pathologist which he, in his turn, cannot interpret. The clinical pathologist, not seeing the case, often cannot speak definitely as to the character of the case judged by the blood changes alone.

The result is that each class of observer has his own definite views regarding the class of anæmias concerned; and the task of endeavouring to correlate the various classes of facts is not an easy one without bringing together in one whole all the data concerned. Nevertheless it seems desirable to essay this task in the hope that clearness may be thrown upon the problems

presented. Such is the object of the present communication. It is based on a very extended analysis of all my records: clinical, pathological, and ætiological, including a very detailed study of the thousand and more blood reports contained in them. My own conclusions based on these data are definite. My difficulty now is how to compress the numerous data on which they have been based, into a form which may serve to make my conclusions clear. The data referred to include those relating to:—

- (1) Types of anæmia concerned and their distinguishing features.
- (2) Character of nervous features presented.
- (3) Clinical features and their relation to the nervous features.
- (4) Character of infective lesions.
- (5) Blood changes.

(b) *Origin of Writer's Interest in the Subject.*

The whole subject of the nervous disorders in anæmia has been of intense interest to me for many years, and has been under my observation and interpretation since the first case of pernicious anæmia I investigated (1889). In that case I first drew attention to nervous features "well fitted to arrest attention" in direct relation to the well-marked features of blood destruction which I then described for the first time, and also in relation to the peculiar sore tongue, which was then described by me for the first time.

This interest was further developed and increased by observations made ten years later (1900), which originated the great subject of "oral sepsis," and brought it into relation not only with this anæmia but with other severe anæmias ("septic anæmia"), and also with nervous disturbances and lesions ("toxic neuritis") which I found to be producible by such chronic sepsis.

Up to that time (1900) the number of cases of pernicious anæmia I had seen had been very few. But in these few cases, ten or twelve in all, I had been struck by the fact that two of them had presented the features of sub-acute combined sclerosis of the cord, and others had shown peripheral nerve disturbance of various kinds (peripheral paræsthesiæ and atrophy of muscles).

When I found (1900) that similar peripheral disturbances, even more marked, could be produced by oral sepsis alone, quite apart from any form of severe anæmia, the question that arose in my mind, as I described it in my work on "Pernicious Anæmia" in 1900, was: Were the nervous symptoms presented in cases of pernicious hæmolytic disease due to the *special hæmolytic* infection and toxin underlying that disease? (A); or were they, in whole or in part, the effects of the *concurrent septic infection (oral sepsis)* which, as I showed, existed and complicated the hæmolytic anæmia?

At first I was inclined to refer them largely to the septic infection rather than to the hæmolytic, since I found what appeared to be precisely similar toxic neuritis produced by extreme oral sepsis, apart altogether from pernicious anæmia—an observation then made for the first time in the history of the subject of sepsis. But I left the matter open: as to how far the hæmolytic toxin might also be largely responsible for these nervous disturbances.

My object is to summarize the results of my observations during the past thirty years in a series of 150 cases of this hæmolytic anæmia and many cases of septic anæmia, observations controlled by post-mortems in forty cases, naked-eye specimens from which are shown in the forty pathological specimens now exhibited. The result shows conclusively that the nervous disorders in the hæmolytic disease—pernicious anæmia—far exceed in their frequency and

TABLE I.—TYPES OF BLOOD CHANGES MET WITH IN THE TWO TYPES OF SEVERE ANÆMIA.

"Type A: "Pernicious anemia" (seasonal, glositic, hæmolytic).—Case 3, complicated by severe oral sepsis and septic anemia. Case 10, case with combined sclerosis. Case 15, freed from oral sepsis and septic anemia.

*Type B: "Septic anemia" (non-seasonal, non-glossitic, non-hæmolytic).* (See also Table IV, p. 35).

Type A: Case 3.—F. H., aged 47.

Year	Date	Red cells	Hemo- globin	Colour index	Leuco- cytes	Poly- morphs	Lympho- cytes	Normo- blasts	Megalo- blasts	Progress	Percentage of iron in dry residue of organs
	Normal	5,000,000	100 per cent.	1.0	7,500	5,000	2,000	0	0		
1st	Onset Sept., 1912	%			%	%	%				
	Jan. 27, 1913	25	30	1.1	40	40	35	14	0	Very ill	
	Feb. 9, 1913	19	24	1.2	48	50	44	20	5	Much better	
	Feb. 22, 1913	18	30	1.6	73	85	55	13	3	Feeling well. Got up. Left hospital April 4, 1913	Glossitic Hamolytic
	March 17, 1913	20	35	1.7	36	28	44	13	0	Resumed work	
2nd	April 29, 1913	56	80	1.4	52	47	56	0	2		
	June 19, 1913	60	72	1.2	—	—	—	—	—		
	July 28, 1913	68	80	1.3	70	60	99	0	0	Very well	
	Nov. 3, 1913	43	46	1.07	39	33	49	5	0	Relapse Oct., 1913	
	Dec. 1, 1913	23	25	1.2	—	—	—	—	—	Very ill. Hospital Death	Liver 2.00 Kidney 0.113 Spleen 0.189
	Dec. 12, 1913	8	12	1.5	11	6	26	1	1		
	Average ...	34	43	1.26	46	43	51	6.8	4/8		

Case 15.—J. H., aged 47.

	Onset July, 1916	—	—	—	—	—	—	—	—	Glossitic Haemolytic
1st	Relapse Sept., 1917	—	—	—	—	—	—	—	—	
2nd	" " 1918	—	—	—	—	—	—	—	—	
3rd	" " 1919	—	—	—	—	—	—	—	—	
4th	Jan. 14, 1920	38	45	1-1	27	24	49	0	0	
	Feb. 18, 1920	67	68	1-0	54	33	105	0	0	
	March 19, 1920	76	74	0-9	84	60	131	0	0	
	May 5, 1920	72	90	1-2	54	27	121	0	0	
										Well.
										"
										"
5th	July 23, 1920	70	88	1-2	57	40	100	0	0	
	March 1, 1921	75	70	0-9	133	150	97	0	0	
	Average ...	66	72	1-1	68	56	104	0.6	0.6	
										Liver 0.076
										Kidney Spleen 0.004 0.030





severity those presented in the only other form of anæmia which presents them, viz., septic anæmia. This fact, along with many others to which I shall refer, appears conclusively to denote that the hæmolytic toxin of that disease is also one that is peculiarly neurotoxic in its action. This is strikingly demonstrated in the actual infective lesions which I find in the tongue (see figs. 1 and 2, pp. 9, 10) in this disease. These lesions show that in immediate relation to the infective foci the most intense neuritic and atrophic muscle changes may be found, changes entirely absent in all other anæmic conditions or other diseases which I have likewise investigated.

Many other facts which I have in review satisfy me that the hæmolytic toxin of the peculiar disease termed "idiopathic pernicious anæmia" (A) is highly specific and selective in its action in relation to the blood, to blood destruction, to blood formation, and in its neurotoxic action on the nervous system.

On the other hand, a comparable series of facts relating to other forms of severe anæmias, notably the form associated with chronic sepsis, which I have differentiated (1900) and termed "septic anæmia" (B), just as clearly show that the "septic toxins" produced by the chronic septic lesions associated with this form of anæmia are equally non-specific in their action on the blood, the blood-forming organs, and in the neurotoxic action which they may, and frequently do, exert on the nervous system. As regards blood destruction they have no action at all.

Interesting as these facts may be, the class of cases of severe anæmias here concerned is excessively small compared with the whole class of other diseases; and the proportion of such cases capable of manifesting nervous disorders of any kind is naturally even smaller. Any conclusions relating to the importance of nervous disorders in such anæmias might therefore be of little importance, if they applied only to such anæmias.

But my personal interest in these anæmias consists in the various classes of facts which I have ascertained regarding their pathogenesis and causation having proved to possess a wide significance in relation not only to anæmias but to nervous disorders of all kinds, ranging from peripheral neuritis up to chronic insanity, and even, as in one of my cases, to acute mania. For the outcome of my studies has been to reveal the existence and importance of two classes of infective lesions underlying them. These are represented by (1) those present in the tongue in the case of the hæmolytic disease, pernicious anæmia (A); and (2) the widely prevalent and, up to 1900, entirely overlooked septic lesions in the mouth associated with septic gums, and teeth, and tooth sockets, and bone around the sockets ("oral sepsis"); also in the nasal sinuses and throat ("nasal sepsis"), also in the stomach ("septic gastritis"), and in the intestine ("septic enteritis and colitis"), all of which may underlie septic anæmia (B).

This latter class of infective lesions, up to 1900 regarded as negligible, have now acquired an importance second to none in medicine; and the outcome in connexion with nervous disorders and insanity is beginning to prove to be of far-reaching and fundamental practical importance in mental disorders.

The outcome in relation to the new glossitic infective lesions which I described in the "sore tongue" of the hæmolytic disease pernicious anæmia (A), is also beginning to be manifest. For now, after thirty years, these tongue lesions—the "sore tongue" of pernicious anæmia—are being spoken of, and their significance confirmed, although in English literature I do not know of a

single article dealing with it apart from my own. But in German medicine<sup>1</sup> a definite literature has arisen since 1910, regarding it under the title of "Hunterian Glossitis," and distinct confirmation given as to its great constancy and pathognomonic and diagnostic significance as one of the earliest distinctive features of the disease. In no relation, however, are these infective tongue lesions more interesting and instructive than in connexion with the nervous manifestations which are always in some degree associated with the disease.

(c) *Toxic Nervous Features in Pernicious Anæmia.*

These are among the commonest and the most interesting group of features presented by the disease.

The toxic nervous features exhibited in cases of the severe hæmolytic disease termed pernicious anæmia (A) occur in relation to its manifestations of sore tongue (or sore stomach or sore bowel) on the one hand, and its evidences of increased blood destruction (hæmolysis) on the other. They are of the greatest clinical importance. They are specially interesting to me, since it was these peculiar toxic nervous features that I described for the first time (1889) in the first case I ever investigated, and brought into direct relation with the features of blood destruction which I then also for the first time described, and also with the sore tongue, which I then also described for the first time. The full account I then gave is contained in "Pernicious Anæmia," 1900, Case 1; also in *British Medical Journal*, 1890, i.

*Case I, 1889: Toræmic Attacks.*—The patient is subject from time to time to peculiar exacerbations of weakness of more or less sudden onset, marked by certain highly characteristic nervous features well fitted to arrest attention, viz., exacerbations of blood destruction, marked by increase of lemon complexion and higher coloured urine, and accompanied by nervous features, intense weakness ushered in by drowsiness as if the patient was under the influence of a slight narcotic, passing off quickly in twenty-four hours and leaving him much weaker. These attacks occurred from time to time, constituting what were termed his "bad days," and they were always accompanied and followed by passage of urine of remarkably high colour, containing urobilin and increase of iron.

These exacerbations presented all the phenomena of toxic poisoning, whether in regard to the more nervous phenomena—the drowsiness, contracted pupils, slight fever and sweating, or the local manifestations of the action of such poison (the intestinal disturbance), or the obviously increased destruction of blood.

All these phenomena were so closely related to each other in their occurrence and their degree, and were always marked by such an increase in the patient's weakness as to point, in my opinion, to the closest possible connexion between them—the weakness being due to an excessive destruction of blood caused by the absorption of specific poisons from the alimentary tract.

That the alimentary tract was the seat of production of these poisons there cannot, I think, be any doubt. The probable seat of formation of the poisons responsible for these symptoms is, however, not the intestinal contents as a whole, but particular lesions in the mucous membrane of that tract—especially in the stomach—which showed evidences of subacute and chronic gastritis, and in the tongue, in which there was a history of soreness as one of the first features of the disease nearly three years before.

<sup>1</sup> "Hunterian Glossitis in Pernicious Anæmia," by Dr. J. Sakheim, *Folia Hæmatologica*, May, 1922. One of the first and happiest results of the glossitic criteria I have established for the hæmolytic disease (glossitic hæmolytic anæmia), known as pernicious anæmia (A), is to show that bothrioccephalus anæmia—which German and many other observers have always regarded as "the most typical form of pernicious anæmia"—has no glossitic features, and therefore is quite distinct from the glossitic hæmolytic disease (A). Its interest has always been that like the latter it is hæmolytic, but as now shown by Schaumann, it is non-glossitic, and therefore not the disease, *sui generis*, which I have termed "glossitic hæmolytic anæmia."

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There was, moreover, a definite history of infection in the case from which the illness dated. I am disposed, therefore, to regard the gastric mucosa as the seat of that infection, the affection of the tongue being probably of the same nature as that of the stomach. Successful infection having once occurred, the history was no longer one of gastric trouble, but one of steadily increasing weakness with all the symptoms we have learnt to regard as characteristic of pernicious anæmia.

The case here referred to has proved historic in its description of all the clinical features which all my subsequent observations have shown to be important, namely, their distinctive character as a whole: the importance and significance of the sore tongue and of the gastro-intestinal features, denoting the site of the disease; of the hæmolytic features, denoting the predominant character of the blood change; and lastly of the toxic nervous features and their relation to the foregoing clinical features.

*A Glossitic, Hæmolytic, Neuropathic Disease.*—Such is the character of this anæmic disease (A) termed pernicious anæmia.

The toxic features described in the above case have also proved to be of historic interest in wider relations. As shown by comments and judgments of many writers, they form the foundation of what has been variously termed "the toxic hypothesis of severe anæmias"; "the doctrine of the enterogenous origin of severe anæmias"; also of more general doctrines subsequently variously designated: "intestinal toxæmia," "intestinal auto-intoxication" or "alimentary toxæmia"—doctrines which in that early period (1890) were unknown, and have since become so prominent.

In one important respect, however, the observations made in this first case and the interpretation put upon them differ notably from most of the subsequent observations and interpretations connected with the general subjects termed "intestinal auto-intoxication" or the still vaguer conditions termed "alimentary toxæmia."

The particular point of difference was, and is, that in these earliest studies my concern was to show that the toxins responsible for the effects described (the hæmolytic and nervous features respectively), were not the result of mere general disturbances in the chemical processes going on in the contents of the alimentary canal—e.g., disturbances connected with indigestion of food constituents, or with putrefactive changes. On the contrary the origin of the toxins was referred back to *particular infective lesions* situated in the mucosa of that tract (e.g., in the tongue, stomach or intestine).

### (d) *Character of Infective Lesions.*

The character of these lesions became from that time (1890) onwards my special subject of interest. Their study in the course of the next ten years (1890-1900) led me onward step by step to the final observations which I recorded, in 1900, regarding the existence of two quite distinct sets of infective lesions in the alimentary tract in anæmias, namely:—

(1) Those connected with, and peculiar to, the hæmolytic anæmic disease (A) termed pernicious anæmia, namely the lesions of its peculiar sore tongue; which I likewise found in the mucosa of the stomach and intestine: lesions which I may briefly term "hæmolytic" (*see* figs. 1, 2, 5, 6, 7).

(2) Those connected with the gums and teeth and sockets, and bone in the mouth, to which I gave the title of "oral sepsis"; these underlay another (non-hæmolytic) form of severe anæmia, which I then differentiated and termed "septic anæmia" (B)—lesions which may be designated as "septic."

In both cases, then, as I have said, the infections underlying these forms of anæmia were traced back to particular lesions and foci, and not merely to vague hypothetical processes of general toxæmia occurring in the alimentary tract.

The result of keeping my attention fixed on these two classes of lesions has been of special interest in connexion with the glossitic lesions of the anæmic disease (A) called pernicious anæmia, and the nervous disorders connected with that disease. For, as I shall show later, I have found every class of disturbance and lesion exhibited by that disease represented in the lesions present in the tongue—viz., pains, paræsthesiæ, degeneration and atrophy.

As regards this peculiar hæmolytic anæmic disease (A), all my subsequent



FIG. 1.—Tongue lesion of "pernicious anæmia" (idiopathic hæmolytic anæmia). The plate shows an infective vesicle between the epithelium and the substance of tongue, with marked neuritic and toxic changes in the muscle fibres beneath.

studies for thirty years have confirmed in fullest measure, and in many unexpected ways, the interpretation I thus put on the features of the first case I investigated. This is particularly the case with regard to the sore tongue I then described.

The clinical aspects, pathological characters, and the epidemiological character of seasonal incidence which I have successively shown to be attributes of this peculiar sore tongue, stamp them as being the most interesting, important, pathognomonic and diagnostic features of the disease. Hence the name I have suggested to connote this disease—"glossitic hæmolytic anæmia," or more shortly "glossitic anæmia."

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They supply an entirely fresh set of criteria in the diagnosis of that anæmia from all others; also new criteria in the interpretation of its apparently manifold, but in reality very well defined, clinical manifestations. These diagnostic criteria in my experience get rid of the difficulties connected with diagnosis by blood changes alone. For these blood changes vary greatly in different cases, and in the same case at different times, in different stages not only of this, but also of forms of secondary anæmia—notably the most common and severe form of all—that which I have differentiated and termed "septic anæmia" (B), so liable at times to be mistaken for the great hæmolytic disease termed pernicious anæmia (A) (see Tables I to IV, pp. 4, 5, 28, 34, 35).

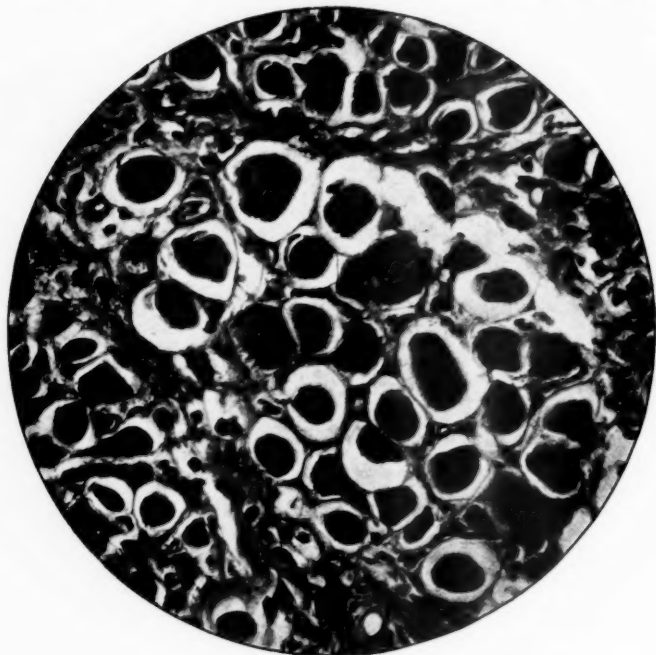


FIG. 2.—Shows the extreme toxic degenerative changes in the muscular bundles of the tongue near the lesion shown in fig. 1. Changes in the nerves of the tongue (neuritis) accompany these lesions.

This help in diagnosis is particularly valuable in the case of the nervous disorders so often associated with one or other of these two forms of anæmia. For one feature of such disorders consists in the degree of anæmia associated with them being often very moderate, e.g., 60 or 70 per cent. of red cells or even higher figures of 80 to 90 per cent.—degrees of anæmia which make it impossible to tell the nature of the anæmia by blood characters alone (see Table V, pp. 36, 37).

This difficulty has been particularly felt in the case of the worst nervous disorders associated with severe anæmia—namely, those denoting sclerosis of the cord—presenting the features of subacute combined sclerosis. The



FIG. 3.—Shows appearance of muscular fibres in the normal tongue (compare with fig. 2).

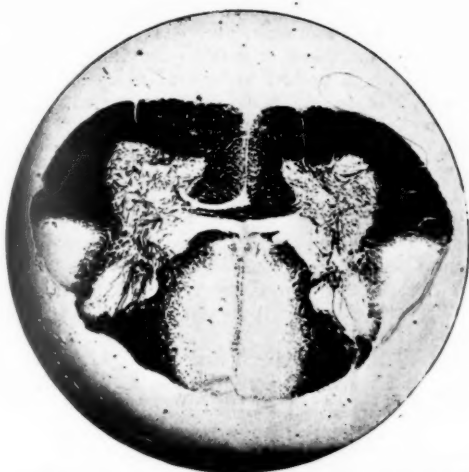


FIG. 4.—Cord from a case of pernicious anæmia (Case 1), showing combined sclerosis (in posterior and lateral columns). The lesions found in this case were very marked in the jejunum (see fig. 7).



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occurrence of sclerosis in anæmias is now well established. The question connected with it is as to the character of the anæmia: is it of the glossitic and hæmolytic type termed pernicious anæmia (A), or may it also be associated with types of secondary anæmia, especially that termed septic anæmia (B)—non-glossitic and non-hæmolytic? (see Table V, pp. 36, 37).

For some twenty years or more that question has been much in my mind, and I now propose to answer it in the account to follow (Section III, p. 26).

### SECTION II.—CHARACTER OF NERVOUS FEATURES IN PERNICIOUS ANÆMIA.

The general nervous features presented in 150 of my cases of this disease fall into five chief groups, and are representative of the classes of nervous features which are most commonly met with in that anæmia.

(a) *Glossitic, gastric, and intestinal pains*, evidenced by soreness connected with tongue, stomach and intestine.

(b) *Toxic*, nervous features related to blood destruction.

(c) *Peripheral paræsthesiæ*, neuritis, and occasionally atrophies.

(d) *Cerebral and mental symptoms*, denoting effects on the higher nerve centres.

(e) *Paralysis, spasticity and ataxia*, denoting sclerosis of cord.

(a) and (b) *The Glossitic and Toxic Nerve Features, and their Relation to one another.*

The general character of these may be illustrated by the features presented, and closely studied by me, in one case which was under my observation for thirteen years (1900-13), after being successfully treated in 1900, and in which the patient—a level-headed, sensible man—spoke to me fully about them.<sup>1</sup> I describe the features of the last relapse he had in 1903, and draw attention to them. They well illustrate the sore tongue and toxic nervous features of pernicious anæmia, namely, the character of the pains connected with the sore tongue, sore stomach, and occasionally sore bowel, and the relation of the lesions in these organs to the hæmolysis, nervous features and degree of blood change presented by the disease.

The facts being as stated in the following record, the question will be raised—as it always is raised in connexion with every class of observation relating to any anæmia bearing the confusing name of “pernicious”—whether the case was one of this kind at all. On this point I will merely say that in all my experience I have never seen a more typical case—so far as all its features were concerned. At the time I first treated it in 1900, I had two other cases under my care in the same ward. One of these patients (Case 3), with the same sore tongue and hæmolytic features, was in the first attack of the disease—some five months' duration—and died ten days after admission. He showed the most intense pigment changes in his liver and kidney, and most striking lesions in his tongue and stomach (represented in my work on “Severest Anæmias,” 1910, in Plates III, figs. 5 and 6; IV, figs. 7 and 8; VII, fig. 13; VIII, figs. 15 and 16; IX, figs. 17 and 18; and X, fig. 19).

The other case was admitted in the third or fourth attack of his disease, made a temporary recovery, but quickly relapsed and died about eight months after first admission. He also had the history of sore tongue, and showed post

<sup>1</sup>Case 10, “Pernicious Anæmia,” 1900; also *Trans. Med. Soc. Lond.*, 1901.



## GASTRIC LESIONS.



FIG. 5.—Mucosa and submucosa of stomach from a case of glossitic hæmolytic (pernicious) anemia, which died in an early stage, and showed the tongue lesions (represented in Plates III and IV of "Severest Anemias," 1910). It shows normal thickness and appearances, the epithelium of the opening of the tubules having fallen out from maceration.

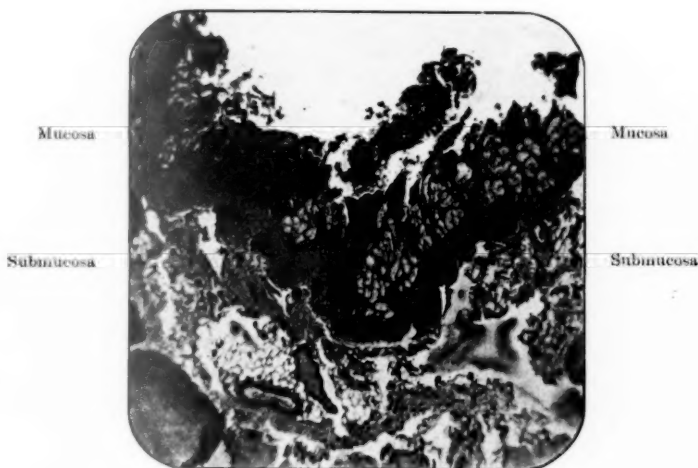


FIG. 6.—Mucosa and submucosa of stomach from the same case as above, from another portion of the surface. It shows intensely inflammatory and ulcerative and necrotic changes—affecting at one point (to the left) the whole of the mucosa; at another part (to the right), glands can still be seen at the lower part of the mucosa. The inflammatory and necrotic exudation shown on the surface, both in this figure and in all other cases, is loaded with streptococcal organisms, and is thrown off as blood-stained and mucoid vomit during life. The histological characters of this vomit are identical with those of the exudation on the mucosa of the stomach, and the streptococci correspond with those found in the tongue.

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mortem the characteristic pigment changes in the liver and kidney found in this disease.

The third case was the one now described (Case 12). He was admitted in the third attack of his disease. He made a strikingly rapid recovery, and even finally recovered, after three years, from his disease, and lived fifteen years longer, as the record I now give shows:—<sup>1</sup>

CASE 12.—PERNICIOUS ANÆMIA: A TYPICAL CASE OF "GLOSSITIC ANÆMIA." (HUNTER) DESCRIBED IN RELATION TO ITS SORE TONGUE AND NERVOUS FEATURES FOR FIVE YEARS, FOLLOWED BY RECOVERY FOR TWENTY YEARS.

July 27, 1900: A man, aged 37. *Illness began two years ago, loss of appetite and weight; lemon colour, and especially periodic acute attacks of pain in his mouth and stomach, the tongue becoming unbearably sore, with big red patches on the dorsum and edges. "The tongue felt as if it had no covering, as if it was quite raw; a piece of bread in my mouth felt like sand paper."* When the attacks were over, *usually lasting two to three days, he could eat beef steak or any other food. The attacks came on almost every three weeks, and were followed or accompanied by gastric symptoms, which from that time onwards became the most prominent complaint—namely, gastric pain, nausea, retching (sore stomach).*

While in hospital, July 4 to August 16, 1900, tongue was sore from time to time. *Excellent recovery.* Red cells rose from 27 per cent. up to 73 per cent.

The case was described and shown at this Society in 1901,<sup>2</sup> and shown again at the Clinical Section of this Society, December, 1910.<sup>3</sup> A relapse occurred in the autumn of 1901; red cells falling from 98 per cent. down to 61 per cent.; followed by rapid recovery to 92 per cent. Another relapse occurred in the autumn of 1902, red cells falling to 50 per cent., followed by recovery to 83 per cent. Another relapse occurred in the autumn of 1903, red cells falling to 30 per cent., followed by striking recovery in three months (red cells 90 per cent. and hæmoglobin 120 per cent.). No subsequent relapses. In December, 1910, red cells 105 per cent., hæmoglobin 115 per cent.; in July, 1913, red cells 84 per cent., hæmoglobin 90 per cent.; leucocytes 9,260. The whole of the blood changes from 1900 to 1913 are shown in Table V, p. 37.

October 4, 1900: He was very well and had put on weight. *Tongue looks red.* He states it has been very sore last four days—"feels cracked all over" when he eats. Last attack was four weeks ago, lasting about a week.

### *Condition of Tongue, 1901-10.*

January, 1901: Dorsum of tongue shows a small suspicious patch of smoothness at the old site; another becomes more prominent on applying a weak carbolic lotion. It stands out red, while the rest of mucosa becomes white.

February, 1901: *Tongue lost its tenderness* two days after last visit. It is now distinctly better. The red patch seen last time no longer becomes red on carbolizing. The fissures also then seen no longer become red. But there is a small irregular red patch behind the old one, and the compound papillæ still show some redness. Health very good. (Hæmoglobin 106 per cent.)

February 22, 1901: Tongue a little tender on tip; patches on dorsum become visible on carbolizing. *Stomach discomfort* this last week, with dull headache. (Hæmoglobin 104 per cent.)

February 28, 1901—March 21, *epigastric discomfort.* Colour of urine higher (hæmolytic); had to stay at home one day. (Reds 90 per cent., hæmoglobin 100 per cent.)

<sup>1</sup> In describing it I have found it necessary to emphasize by italics the particular class of feature occurring from time to time to which importance is to be attached. I cannot in any other way than by this over-italicizing bring out what I desire to convey to the reader.

<sup>2</sup> *Med. Chir. Trans.*, 1901, lxxxiv, pp. 203-249.

<sup>3</sup> *Proc. Roy. Soc. Med.*, 1910-11, iv (Clin. Sect.), p. 37.

## INTESTINAL LESIONS.

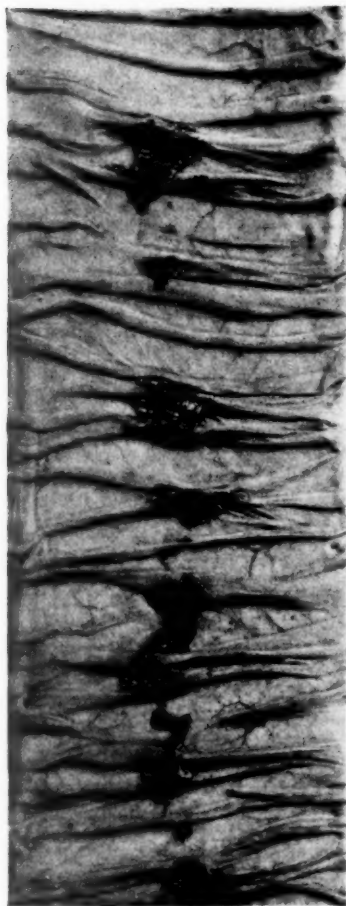


FIG. 7.—Lesions shown in jejunum in a case of glossitic hæmolytic (pernicious) anæmia with marked cord symptoms (combined degeneration). (Case 7.) Patches of croupous enteritis are here shown, situated on or near the valvulae. If the larger ones were not present, the smallest might readily escape notice, especially if, as is often the case, the intestine is washed out with a strong stream of water before it is opened or examined. In the course of over 1,000 post-mortems made by me in which I kept closest observation on conditions in the intestine, I never met with lesions exactly similar to or comparable with the above. But in several cases of pernicious anæmia I have observed changes of redness and patches on the valvulae of the jejunum which have seemed to be suspiciously like those presented in certain parts of the above lesions.

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August 17, 1901: Last seen two months ago. Last fortnight *mouth bad*: getting worse past week; *tongue sore and patchy* behind; sore at root of tongue. *Stomach* most *uncomfortable*, very *sick* one day last week, no appetite, no taste in mouth. Tongue shows no patch and no redness, but fauces much congested, and anterior surface of soft palate and *pillars of fauces* covered with minute pin-head *translucent-looking granulations*. Tongue so sore four days ago that he could hardly do anything. After carbolicizing, angry red patches on dorsum, and angry red granulations on right edges and tip of tongue. "This is the second attack of sore mouth he has recently had; usually one every three or four weeks." (Red cells 63 per cent., hæmoglobin 80 per cent. *First relapse*.)

August 27, 1901: Looking much better; redness all gone from palate; tongue shows nothing abnormal.

November 22, 1901: Very well since last seen. Still some *stomach discomfort*, but taking food well. *Tongue* has not been sore since; it is remarkably clean and *healthy looking*; good colour; not smooth; without any redness. (Red cells 62 per cent., hæmoglobin 84 per cent. The blood looks of good colour, and I expected it to be normal from its consistency.)

December 24, 1901: Looks very stout, weight 11 st. 11 lb. *Tongue very clean*. (Red cells 64 per cent., hæmoglobin 75 per cent.)

February 22, 1902: *Looking very well*. Weight 11 st. 13 lb. Mouth and teeth very clean. *Tongue tends to be sore at times*, but always checked by carbolic lotion. (Red cells 92 per cent., hæmoglobin 98 per cent. Good recovery.)

July 28, 1902: Well till a month ago, when he *lost appetite*, and felt nausea; feels "trembly" in his legs, with "gnawing" pains in muscles and bones of legs, especially last ten days. (Peripheral nervous features.) Weight 11 st. 9 lb. Looks stout and well. *Tongue* very clean; sometimes *slightly tender*. (Red cells 70 per cent., hæmoglobin 100 per cent. Commencement of second relapse.)

November 3, 1902: Well till a week ago, since then *dreadfully tired and depressed*. Tongue very clean, but it has been *sensitive this last week*. Nothing visibly wrong on it. Appetite good. Has had *pains in his back and legs ache* from knees downwards, and *fingers still go numb* when he is in bed. He looks stout, but he has a *slight lemon colour of conjunctivæ* (*hæmolytic features*), and urine shows marked *urobilinuria*. (Red cells 50 per cent., hæmoglobin 60 per cent. *Relapse more marked*.)

November 17, 1902: Feeling better, but much troubled with sleeplessness, and aching in legs; he wakes up very tired in the morning. *Colour rather lemon*. Tongue very clean.

### *Relapse with well-marked Nervous Features.*

April 27, 1903: In March he had felt wonderfully well, as if all his strength had come back to him. Suddenly, about April 20, he became worse, felt as if he had been knocked down and kicked from head to foot, as if he had been bruised. He was sore whichever way he turned. All last week his head was "right distracted with the least thing"; everything seemed to worry him to a degree that he did not know where he was. If anyone speaks, he cannot hear them because of his head. He feels dreadfully depressed. His wife complains that he is "so disagreeable"; but at all these times (i.e., when his sore tongue returns) when his attacks are on, he simply cannot help being irritable and disagreeable. His mouth has been sore this last week, and it is sore now. When walking he feels as if he had lost all his strength up to the hips; the strength goes out of his back; he feels as if he cannot pick his feet off the ground, and goes with them sliding along. He has often thought people might "think him drunk," and he feels dazed and lost. At these times he cannot walk without a stick. It is when he is at the top of steps that he feels most nervous, he has to hold on to banisters to come down, especially when he looks. At business no one notices his irritability, but on one or two occasions he had felt so overdone that a friend thought he had had some drink, he was so dazed. He likes to be by himself, and cannot bear even his children to talk. Indoors, when speaking, he often gets very excited and irritable. At present his hands are numb, especially at night, and feel as if electricity was trickling

all through them. There is often cramp in calves and legs at night. Tongue looks clean, but he states it is slightly tender; no red spots to be seen on it.

All this account came from a man of apparently placid disposition, looking stout and well. Yet his blood on examination showed only 74 per cent. of red cells, with colour index 1.2. And from this time onward his attack developed, and I draw attention to the character of the nervous features, and their relation to the infective lesions of his disease as represented by his sore tongue.

May 11, 1903: He was "bad" all last week, so tired, "head bad," as if it was not his head at all; everything worries him to death. From knees downwards his legs seem to get on fire. His tongue looks absolutely normal and clean, but he has some pains in his stomach. His weight has fallen about 10 lb., viz., down to 11 st. 2½ lb.

July 10, 1903: He went on well for three or four weeks after being last seen. Then the mouth "got bad," the tongue sore (June 11) for a fortnight and then got better. But last week two or three sore places broke out, and he felt "depressed and altogether wrong." Tongue now improving, not sore. It is quite clean, but shows a red spot on right edge. He has felt very sick without any appetite these last three days (sore stomach). He has still numbness and tingling in fingers, some days more than others. His legs ache at night from knees downwards giving him no rest, and they are equally bad when he wakes in the morning (nervous features). (Red cells 60 per cent., colour index 1.4; anæmia increasing.)

September 9, 1903: He got better after being last seen, but in last week of July mouth became sore, and was found to be very ulcerated; every part of it sore, gums, roof and throat and tongue also very bad, worse than at any time since his illness began—at the end of 1898. This continued for a fortnight. He got very weak, lost all his strength, no sickness. He went away for two weeks, and then returned and went back to business, but felt very weak, and then after a week had to give up work and he had one very bad day of diarrhoea (sore bowel). The last week in August he gradually picked up his strength. Weight 11 st. He does not look so stout. Conjunctive slightly lemon coloured (hæmolytic features). Tongue now quite clean, but shows considerable change since last seen. There is now a large smooth area on the dorsum, where formerly there were healthy papillæ. It is not tender now, but a few weeks ago it was so sore that even speaking hurt him, and he was "as yellow as possible" at the time. ("Glossitic hæmolytic anæmia.") Red cells 45 per cent., colour index 1.3—a further fall.

The features of this attack are of particular interest. They were under my immediate observation. They represent exactly the first features of the disease as they usually occur—a sore tongue, followed by hæmolysis, anæmia and weakness. The patient was stout and of good colour. There was nothing in his appearance to suggest that he had any anæmia, least of all a degree of anæmia so marked as 45 per cent. But his sore tongue had been very marked, as also his hæmolysis shown by lemon colour and very high coloured urine with urobilin (specimen now shown). The anæmia had increased, and the nervous features at the onset had been very severe.

October 23, 1903: In September he felt better, except that he was troubled with pains in loins and back, and he continued well till October 17, when everything he took gave him pain in his stomach (sore stomach), with nausea, but no actual sickness: tongue began to get sore on October 18, cracks began to form, and then on October 19 he suddenly got very yellow (sudden onset of hæmolysis). Tongue is still a little sore, but not so bad as before. He feels "terribly weak"; his head aches from back of eyes and over top of head, so that he feels quite dazed, and hardly knows sometimes what he says ("head bad"—nervous features). He looks thinner and paler; weight 10 st. 8 lb., loss of 7 lb. The tongue looks clean, and shows no obvious cracks or abrasions, but it is very tender on left edge. (Blood shows: Red cells 31 per cent., colour index 1.6—a further deterioration.)

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Here again there was nothing in his colour or appearance to suggest that he had such a degree of blood change. It was almost by accident that I made a blood examination when I saw him.

November 2, 1903: His mouth had got sore again, with red patches on roof, similar to those on tongue, so sore that he could only take milk. There was no actual sickness. There was looseness of bowels (three times this morning). ("Sore tongue, sore stomach, sore bowel.") In spite of these he thinks he feels better than last week. His tongue is red; quite clean and glossy over a very considerable area; a little sore when taking food, but not so bad as last week. Mucosa of mouth normal. No trace of oral sepsis. (Blood: Red cells 46 per cent., colour index 1'3—an improvement of 15 per cent.)

This proved to be the last attack the patient ever had. He went on free from his disease from that time, 1903, till his death in 1918 from another disease—a period of twenty years from his first attack in 1898 and eighteen years from the date of his first treatment by me in 1900, and fifteen years from date of his last attack in 1903.

I saw him from time to time between 1903 and 1913. He looked the picture of health—e.g., July, 1905: Stout, ruddy, weight 12 st. 5 lb.; appetite good; bowels regular; no nervous feelings; tongue absolutely normal; no trace of glossiness. Blood: red cells 91 per cent., with colour index 1'3. December, 1913: Red cells 106 per cent., colour index 1'1.

### *Commentary.*

The case above summarized is in all its features the most interesting I have observed in the course of my thirty-two years' studies of the disease. It is only the second case I have recorded separately in the course of these studies. In the first one, recorded in 1890, there was described for the first time a series of twenty-one clinical features as distinguishing the disease—the teaching up to that time being that it had no distinctive clinical features. This second case was first recorded by me in my work on "Pernicious Anæmia" in 1900; and afterwards separately, with a commentary, in the *Medico-Chirurgical Transactions*, in 1901. It was the first case I treated on the antiseptic lines which I recommended in 1900, as the result of my studies revealing the close and intimate association of this hæmolytic disease with sepsis—in the mouth, stomach and intestines ("oral sepsis," "septic gastritis," and "septic enteritis," as I termed these conditions respectively).

In this case (1900) I determined to apply all the principles of treatment I then laid down, namely, the removal and control of all infective lesions presented by the mouth in such cases: (1) The "sore-tongue" lesions (glossitis) which since 1890 I had found to be peculiar to this hæmolytic disease; and (2) the common septic lesions of the teeth, sockets and gums ("oral sepsis," as I termed them) which from 1890 onwards I found to be so frequently associated with this disease *not* as its *causa vera*, but as an intense septic complication.

I, therefore, treated this case in 1900 in a new way by strictest oral antisepsis supplemented by anti-streptococcal serum. The recovery was far more striking than anything I had ever seen before. But the chief problem remaining was how far this recovery would be permanent, and not be interrupted by the relapses with the fatal results so usually observed in the disease. So I resolved to keep the case under my closest watch from week to week, or month to month, as the case might need; and I asked the patient—a most intelligent man, a commercial traveller—to call on me at any time



at my home address whenever he felt anything the matter with him. The particular guide I gave him as to anything being the matter was the return of any symptoms of his sore tongue. He did so at short intervals during the following three years (1900-3) and afterwards at longer intervals during the next ten years (1903-13).

During the first three years, I saw him going through the initial stages of three relapses (see Table V for blood changes), from all of which he recovered without the necessity of lying up. He then recovered permanently, and remained free from his anæmic disease till he died in 1918, as already stated, from some other disease. As the result of this close observation I was able to see not only once but again and again the whole features of the disease and their relation to each other, as they actually occur in every case before the patient comes under the observation of the doctor. This period in the history of each case is on an average about two years before the patient comes under observation.

These observations of the earlier features of the relapses—as regards the character of the “sore tongue,” “sore stomach,” “sore bowel,” “sore hæmolytic,” “sore nervous system,” followed by “sore anæmia”—give a picture of this disease totally different from any presented by the patient after he comes under observation with his disease fully developed. The chief feature of that picture is, that clinical features are all in the foreground, whereas the blood changes are all in the background. The chief feature of the clinical picture in its turn is the varying degree in which the individual clinical features present themselves in different attacks, and in different stages of the disease. The most instructive feature, however, is always the sore tongue; and it is of particular importance because the changes here can be seen and studied.

But apart from these glossitic features marking the varying progress and activity of the disease from time to time, the most interesting and instructive aspects of the case were those connected with the nervous system in the third and worse relapse the patient had at the end of 1903.

*The nervous phenomena* then presented, and described by the patient, were of a most instructive character. They were, as it appeared, far in excess of the degree of his illness (at the time indeed he looked robust, healthy and of excellent colour). Presented by themselves in any similar patient, they would never have suggested any anæmia, or blood change of any kind. They were so intense as to appear almost neurotic or neurasthenic in their character. And yet as the sequel showed, they were not of that character. The blood examination revealed a degree of blood change perfectly astonishing—e.g., a fall to 25 per cent. of red cells in the course of a few weeks in a patient looking apparently robust (10 st. 12 lb. weight), and of good healthy colour.

The description given of his nervous feelings, of his dazed condition from time to time, of his dislike of anyone being with him, of his “head being bad,” of his being unable to walk without tottering or without the aid of a stick, of his suspicions as to what people were thinking of him and what they actually thought of him: all this class of features were not imaginary; they really represent what actually occurs in this disease, and what I have observed from time to time. Little wonder that the pernicious anæmia patient is peculiarly difficult to manage; little wonder that doctors who have had one case under their care for any time have stated to me that they never desire to have another; and little wonder that the cases I have had under my care in private—comparatively few in number in relation to other diseases—have given me more



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trouble and concern and have impressed themselves more on my memory than all the other cases I have ever seen.

Interesting as these nervous features were, it is not on their account that the case is now recorded. It is on account of the *sore-tongue features*—the glossitic features—of this “glossitic anæmia,” as I now after full consideration term the disease. My object is to show how the disease can be recognized even within a few weeks of its first contraction, instead of a year or two later, as is now generally the case; how careful watch on this sore tongue enables me to recognize the recrudescence of activity of the disease at each successive period of the disease, and to take appropriate measures for controlling it at each stage.

### (c) *Peripheral Nervous Features.*

These are of the nature of paræsthesiæ connected with the upper and lower limbs, with the hands and fingers, legs and feet. They are extremely common; and so characteristic that for twenty years and more I have been accustomed to attach a definite diagnostic significance to their presence or absence. Their commonest form is that of feelings of numbness and tingling in hands or feet, arms or legs, accompanied by other feelings which the patient has difficulty in describing—feelings of coldness; as if wind was passing along the legs; as if his skin had been rubbed with sandpaper; as if he had been beaten all over; “trembly” in the legs, with gnawing pains in muscles and bones of legs; aching of legs from knees downwards with pains in the back; numbness of fingers so that he cannot pick up small articles or button his clothes; aching in limbs so that he wakens up very tired in the morning; or with a feeling as if he had been knocked down and kicked from head to foot—as if he had been bruised; numbness in hands, especially at night; also in the feet, as if electricity was trickling all through them, with cramps in calves of legs at night; feelings from knees downwards, as if his legs were on fire; legs aching at night from knees downwards, giving him no rest; and equally bad on awaking in the morning; legs feeling “as if he could not bear the one on the other”; feelings of constriction as if the trousers were too tight.

These symptoms are not constant, but come and go according to the activity of the disease. When close observation is kept on their relation to the other features of the disease an interesting feature of them is that their variations are always related to disturbances connected with the lesions in the tongue, stomach or intestine—namely, with sore tongue, or gastric disturbances, or intestinal trouble (e.g., looseness and discomfort in bowels).

These subjective feelings are sometimes accompanied by hypersensitiveness in legs, from knees downwards, or tenderness of muscles, and by blunting of sensibility to touch; so that, as already stated, the patient has difficulty in picking up small objects, in tying his tie, or buttoning his clothes.

In most cases there is marked loss of vibratory sense, or this is absent in lower limbs while normal in upper limbs; cutaneous sensibility little if at all affected, and no muscular atrophy in any of the cases. In the first case in which there was marked paralysis there was a zone of hyperæsthesia around the abdomen just below the umbilicus.

### (d) *Cerebral and Mental Nervous Features.*

Associated with the foregoing groups of features—toxic and peripheral—many cases present certain features, not so obvious, and only ascertainable when the patient is under observation over a long period and often only from friends and relations in contact with him.

These are connected with neurasthenia and changes in disposition, sometimes very slight, sometimes very marked, that cause great difficulties in the management of the case. These features sometimes come on gradually, but become more pronounced after the first severe attack of his disease. The most prominent of these features is mental depression, "horrible depression," "melancholia," which were the first features in two of the cases (Cases 8 and 10); intense nervous depression, regarded as neurasthenia in a third case (Case 9); these features appearing before any question of anæmia had been raised.

In certain cases (e.g., Case 8) this is followed by a change in disposition; the patient becoming very suspicious of all around him; very secretive about his affairs; changing his bank account lest his relations should know about his affairs; imagining that everyone is discussing his illness and speculating as to who will take his place. These suspicions sometimes take curious forms with startling results, as when one patient sent to a particular home by his own doctor (No. 1), went instead to a second doctor (No. 2) without saying anything about No. 1; was placed by him in another home under another doctor (No. 3); where he was found by the original doctor (No. 1); who then called in another doctor (No. 4).

In the case of another, the patient was sent to convalesce at the seaside and wrote his doctor that he was going on well. He was really lying desperately ill unattended in a top room of a boarding house, and had called in a local surgeon and insisted on being operated on for cancer of the stomach (which of course was not present) and died a month later. In another under my care in a home, the patient had a history of attacks of melancholia, but was very quiet and natural. Suddenly one night he became wildly maniacal and was only with difficulty restrained by two nurses from throwing himself out of the window. Again, in another case recently under my care in hospital, the patient who had a sharp toxic attack, for four or five days, from which he made a quick recovery and without apparently any change in his disposition. But a week later he was found to have written to the police stating that he was being kept in the hospital against his will, although at the time he had been so ill that he lay for several days in a semi-conscious condition passing his stools and urine under him. As I have said, changes of this character are not often spoken of by the patient himself, but when seen, as I have often had occasion to note, they form a remarkable nervous feature of the disease pernicious anæmia.

*Toxæmic Attacks.*—The character of these attacks, as experienced by the patient, is described in the following terms by a patient, an eminent member of the profession, whom I saw early in June, 1900. The case was a very severe and typical case of the disease, in sudden onset, character of sore tongue, sore stomach, sore hæmolytic, sore anæmia, and sore nervous system; also typical in the view the patient took of his case.

"The blood is the only thing in my case that seems to show the disease. I am reduced to the vanishing point in blood, hence my digestive organs are extremely weak. There is nothing else the matter with them. I wish I could make some of you see that with me at any rate the illness is *cardiac*. Hence my heart gets daily worse and I am slowly but surely dying.

Eight physicians have I seen in this bout in about five months and they all tell me quite different. Need you wonder that I am a sceptic of the sceptics? It is a complex disease."

The distressing features in the first week after I saw him were the cardiac weakness, the "sore stomach" (discomfort, nausea, sinking feelings accom-

panied by much flatulence from stomach); pain going down to the tips of both hands and fingers, especially severe in the left; great numbness in soles of feet. "I never felt anything like the mental depression all through." Along with these there was rapid loss of strength: great increase of pallor, especially of mucous membranes; greater frequency and prolonged attacks of heart pain.

During the next three weeks he went through a bad period, which he subsequently (July 9) described as follows—a typical severe toxæmic attack, through which patients pass, and afterwards have little or no recollection of what happened, and from which they make sudden and most remarkable recoveries:—

"My first attempt at writing since I sent my report to you. My own knowledge is not very clear, but I know that I plunged rapidly down, and for days lay in a miserable condition with frequent severe attacks of angina, and only relieved when death appeared imminent. I know several nights I lay with labouring heart, and cold sweat pouring off me, and the agony in both arms to my finger tips, praying for death which would not come, although sometimes my heart seemed to stagger and stop, and I lost consciousness, only to find myself presently struggling back to life. After about a week in an apparently dying condition I began slowly to mend. During my worst time the temperature was between 99°6' F. and 100°2' F. and my pulse varied from 96 to 120, frequently irregular and intermittent, the heart's action always distressing to me. Now (July 9) I am able to be in my chair again for two or three hours in the afternoon; increasing strength; less flatulence."

He made a great recovery, was up and about again a few weeks later, and remained well for five or six years later, when I last saw him in robust health. In a note about a year later (March 3, 1901):—

"I am, and have been for some time (months), to all outward appearance well, better in appearance, colour, &c., than I have been for years, but I am conscious myself of a great change since my illness. To begin with, I never lose the *numbness and tightness and pain on pressure in my feet*—just now they are so bad I can hardly walk at all, and I still have *numb and more or less painful sensations in my hands and arms*. I suffer also from great *stiffness in my muscles*, so that often I can hardly stoop to pick up anything. I have no oral symptoms. I think that I have once or twice had some slight soreness of the tongue. I have no alimentary canal symptoms. I never feel sick, and always have an excellent appetite: could eat much more than I do. I do a great deal of outdoor work. My last blood examination some months ago was nearly 4,000,000 reds with 80 per cent. of hæmoglobin. I have entirely lost my low spirits, though not the reasons for them; worries continue but they are faced with my usual elasticity. I take as much interest in life as before, and in things and people; but I look at everything from an entirely new standpoint, since I spent those weary hours looking directly into the waters of the dark river, and wondering as to the other side. I no longer long to cross, as I often did then, but on the contrary am anxious to continue here for a bit at any rate."

A year and a half later (November 4, 1902) he wrote:—

"I am very fairly well; have been out partridge shooting occasionally this season, and usually work several hours a day out of doors. I have fitted up a carpenter's shop for winter, with a lathe, as I must have lots of exercise. I have ups and downs, and cannot do without the arsenic for long. I got rather anæmic once in the summer, but am better again. So long as I keep quiet at home I am all right."

#### (e) *Features of Sclerosis of Cord.*

In some cases (ten out of my 150 cases) the nervous features include weakness of limbs, paralysis, spasticity and ataxia in varying degrees, denoting some degree or other of sclerosis of the cord, and in the only

case in which a post-mortem was obtained, the cord presented marked lesions of combined degeneration of the lateral and posterior columns. (Fig. 7, p. 15.)

*Case 7.*—Onset, October, 1900. The features in this case included (March, 1901) marked paralysis, with spastic gait and ataxia with increase of knee-jerks, tenderness of legs on pressure, a zone of hyperæsthesia around abdomen, just below umbilicus; some difficulty in defæcation and loss of power over rectum; slight weakness and loss of muscular co-ordination in arms. (For commentary see Section IV, p. 39.)

*Case 8.*—M. Onset of disease, September, 1906. At first (April, 1908), numbness and tingling in both hands and tips of fingers, with blunting of sensibility to touch in both hands, also of feelings on constriction in his legs, as if his trousers were too tight. Then in November, 1908, knee-jerks diminished, but no loss of sensibility to touch, no ataxia; knee-reflexes present, but diminished (profound anæmia, red cells 27 per cent., colour index 0·85, followed by remarkable recovery to 72 per cent., with colour index 0·9 in four weeks, and then to 99 per cent., with colour index 0·8 by February, 1909).

Then in August, 1909, wildly ataxic, scarcely able to stand, and almost falling when he turned; knee-jerks brisk, ankle-jerks absent in left foot; brisk in right, plantars showed flexor response. No cutaneous anæsthesia or analgesia but vibration sense lost in lower limbs, normal in upper limbs. No muscular atrophy, and joint sense normal in upper and lower limbs. For some months his legs had felt stiff, and for the last month had been very tottering. No lightning pains, no diplopia, no bladder trouble, speech and articulation normal, pupils small, reacting sluggishly to light, but briskly to accommodation, no ataxia of upper limbs. Cerebro-spinal fluid not under pressure, containing three cells per cubic centimetre, large and small mononuclears in equal number. Noguchi's test negative. Condition of cerebro-spinal fluid excluded tabes (Dr. J. Purves Stewart, 1909). (Blood at this time: Red cells 84, with colour index 1·07.)

During next two months these features lessened to such a degree that he was able to stand and walk, and his gait much steadier, but blood condition fell to 36 per cent. red cells, with colour index 1·2. Died a month later, or about five months after onset of ataxia.

*Case 9.*—F. Onset, August, 1910. Extreme spasticity and ataxia and trouble in passing urine (March, 1911), unable to stand or walk, marked peripheral pains, knee reflexes increased, ankle clonus present, loss of sensation in bladder (urine had to be drawn off by catheter); loss of sensation in various parts of the body, especially in the finger tips, loss of the muscular sense, spastic contracture of the limbs. She had no idea as to the position of her limbs, and the legs became crossed over each other with such strength that it became very difficult to empty the bladder by catheter.

These conditions continued for some four years, when she died. By means of walking chairs and carrying chairs she was able during this period to spend a good deal of time out of doors or to take occasional carriage or motor drives; she was of good colour and the blood count remained nearly normal till the final illness.

*Case 10.*—M. Onset, August, 1909. Earlier features (February, 1912), of numbness and hypersensitiveness in legs from knees downwards, tingling and numbness in fingers and hands, numbness with tenderness of knees and ankles and aching of the leg bones, feelings of coldness and tingling of knees and legs; knee-jerks increased on both sides; no ankle clonus, loss of vibratory sense (blood showed red cells 43 per cent., with colour index 1·2).

A year later (April, 1913), weakness in limbs and inability to control limbs, especially legs, tingling in fingers, and a general feeling of deadness all over his body. These symptoms had developed about four months before (December, 1912). Unable to walk properly or control movements. Could move legs, but had stiffness and some ataxia, knee-reflexes distinctly increased, and ankle clonus well marked on both sides. Unable to stand erect, muscles very weak and gait very ataxic; could not control legs even

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when supported. General numb feeling in legs, and some sensitiveness when he put them together or crossed his knees. Sensibility to touch present, but slightly impaired to heat, not impaired to cold; impaired to vibration (blood showed red cells 61 per cent., with colour index 1·3).

Left hospital, July, 1913, with much better colour, but with nervous features much the same. Had lost all pains in legs and arms, but had marked weakness of legs with inco-ordination, marked exaggeration of all deep reflexes, and some slight paræsthesiæ in hands and numbness in feet.

Three months later (October, 1913), returned with recurrence of old symptoms, numbness about knees, dull appreciation of light touch, and confusion regarding feeling the point or the head of a pin below level of umbilicus. About level of umbilicus an indefinite zone of diminished sensibility to touch and pain. Below this an area slightly hyperæsthetic; knee-jerks absent left side, slight response right side. Ankle clonus not obtained; Babinski's sign present on both sides; epigastric reflexes present, abdominal and cremasteric not obtained. Deep reflexes in both arms much exaggerated; very slight loss of control of bladder and rectum. Trophic wasting of muscles, especially quadriceps on both sides; motor voluntary movements of legs complete, but no strength in movements. Inco-ordination slight; no power to stand upright or walk (blood showed red cells 61 per cent., with colour index 1·04).

(For course and blood changes of these cases see Tables I, p. 5, and V, p. 36.)

The proportion of cases of the disease which show these changes is small in my experience. In 150 cases of which I have records, about 100 in private cases and about fifty in hospital cases, I have seen marked instances of subacute combined degeneration in ten cases, eight private cases and two hospital cases. This represents approximately about 7 per cent. of all cases. Possibly the number may be considerably larger, since cases of this character come more within the observation of the neurologist than within that of the general physician. On the other hand the proportion of cases seen by neurologists among cases of anæmia may equally be too large, since the cases of anæmia that come under their notice are mostly those presenting severe nervous features.

In only six out of the cases had I the opportunity of obtaining full notes or of keeping observation on them for any length of time. The most important point about them was that they all corresponded in their clinical features, pathology and course with the definite hæmolytic disease termed pernicious anæmia (A), and I have never observed a case of this kind in septic anæmia or any other form of secondary anæmia.

This fact is interesting, since peripheral paræsthesias may in my observation occur in septic anæmia. Indeed a definite form of toxic neuritis, affecting peripheral nerves of arms and shoulders and causing atrophy of muscles in these parts, is in my observation, as originally described in 1900 ("Pernicious Anæmia," 1900) by no means uncommon as an accompaniment and result of extreme oral sepsis, and many obscure forms of neuritis—termed generally "rheumatic"—are in my opinion of a similar character.

Moreover, an even more common accompaniment of chronic sepsis, both oral and nasal, is, I have observed, the occurrence of general nervous disturbance in the cerebral system, shown by neurasthenia, mental depression, melancholia, and general nervous and other features of mental degeneration and insanity (a group of features also represented among those met with in pernicious hæmolytic anæmia).

Such being the case, one might naturally expect that in some cases this chronic sepsis might also produce cord lesions, such as those of subacute combined sclerosis. But as I have stated, I have never met with any case

of this latter character in septic anæmia (B). I have never failed to find that its characters at one time or other were those of the pernicious hæmolytic disease (A), however quiescent the features of the latter disease might in course of time have become—even up to the point of complete arrest and control. This conclusion is an important one in connexion with the pathogenesis of this latter form of sclerosis, for it would indicate that, if producible only by the specific hæmolytic toxin responsible for pernicious anæmia, this form of sclerosis—combined sclerosis—would itself prove to be the result of a definite specific infection having neurotoxic characters—just as the peculiar character of the sclerosis in tabes owes its origin to the action of the specific neurotoxic poison produced by syphilis.

On the point on which I have just touched—as to the unity or plurality of the condition termed “subacute combined sclerosis,” my clinical experience of the latter condition does not enable me to speak, as I have only seen the cases that are associated with anæmia. But the matter is an interesting one; and I give a clue from my clinical experience which may be of interest and help in elucidating the point. The clue is this: That during the past ten to twelve years cases of the pernicious anæmic disease (A) are far more commonly seen than they used to be before 1910. In my experience, it was fortunate if one or two cases could be found in a hospital at any one time. Now, it is quite common to find three or four or as many as seven. It is not uncommon indeed to find two or even three cases in a single ward; and I have frequently had this number under my care. This is not due to any increase in the number of cases of this disease. It is in my judgment connected with the fact, known to me from my experience, that this disease now runs a much milder and more chronic course than it formerly did, and therefore many chronic cases remain alive, and go in and out of hospitals during their various relapses. This change dates from about 1910, and it coincides with the increasing attention now given, from 1910 onwards as I know from the reports of my cases, to removal of sepsis from the mouth in all cases of severe anæmia, as I originally urged in 1900.

If, then, subacute combined sclerosis is connected specially with this form of anæmia, and is the manifestation of the neurotoxic action of the hæmolytic toxin of this anæmic disease, one result of this might be expected to be that cases of subacute combined sclerosis should also be more common in our hospitals than they used to be ten or fifteen years ago. Evidence of this could be obtained from hospital records, preceding and subsequent to the year 1910, by which date the practice of oral antisepsis, introduced in 1900, had become much more general.

Leaving that point out of discussion for the present, the interesting feature of the five definite cases presenting the features of combined degeneration of the cord which I have been able to study is that I was fortunate enough to find them at every stage of the disease, from its onset up to a period of over ten years, and to see the characters of its clinical features and blood changes at these different stages.

The relation of these cases to the hæmolytic anæmic disease (termed “pernicious anæmia”) I shall now bring out in a striking way in the following section, showing how exactly the clinical features and course of the cases of anæmia marked by subacute combined sclerosis correspond, as regards their seasonal incidence and variations, their clinical features (glossitic and hæmolytic) and their blood changes, with those presented by that disease. (See p. 29.)



SECTION III.—THE NATURE OF THE ANÆMIA WITH WHICH COMBINED SCLEROSIS IS ASSOCIATED AS JUDGED BY (a) SEASONAL INCIDENCE, (b) GLOSSITIC AND HÆMOLYTIC FEATURES, (c) BLOOD CHANGES.

- (a) *Individuality of Glossitic Hæmolytic Anæmia ("Pernicious Anæmia") as shown by its Seasonal Incidence and Seasonal Variations (Table II).*

The greatest feature, which crowns as it were the whole superstructure of clinical and pathological features of this disease, is that of its *seasonal incidence and seasonal recurrences*—end of summer and autumn (July, August, September and October), with remissions in early spring and summer (February, March, April, May, June). This great feature, which I reported for the first time in 1913 before the International Congress of Medicine in London, had been under my close observation for the preceding ten years, and I had waited till the number of my cases (150) was large enough to afford sufficient data.

The character of the seasonal variations presented by these 150 cases can be judged from the curves shown in the twenty-two cases shown in Table II (p. 28). The uniformity of incidence, sudden in character in autumn, is very strikingly shown, as also the seasonal recurrence and relapses at the same period. The remissions between February and July are equally striking. The seasonal incidence or periodicity of this disease forms the last and the most remarkable feature which I have ascertained regarding this great glossitic and hæmolytic disease.

I was led to recognize it by the following circumstances, as described by me in 1913:—

(a) The first of these was the frequent mention by patients in case after case of their having been seriously ill at or about Christmas, and of their illness having dated from a period of two or three months before that period.

(b) The next circumstance was the frequent mention of the illness having commenced in the late summer or early autumn (July to September), in many cases connected with a history of some definite exposure to insanitary influences.

(c) I then began to make a record of the state of health of all my patients month by month during the whole course of the disease from the onset. This brought out in a most striking way that the incidence of the disease and the recurrence of it were always connected with the months of August, September or October. The incidence was in the late summer or early autumn, and the recurrence was also about the same period.

(d) Lastly, the circumstance which finally settled the fact of this seasonal incidence was that during the past years when I have perpetually had a number of cases in various stages of the disease, the patients have always undergone some relapse during that above-mentioned period of the year.

So remarkable is this periodicity that I have again and again had patients returning to me reporting their relapses in the same week or fortnight in the early autumn after having been to my knowledge on a high level of recovery (80 to 100 per cent. of red corpuscles) throughout the previous summer.

The periodicity in the recovery is equally striking, namely the fact that the patients who have passed through serious illnesses during the winter months generally have a marked period of recovery and betterment during the months of March, April, May and June.

There is no doubt about the facts which I now report. They are established by the records in every one out of the series of 150 cases.



*Specificity.*—Taken in conjunction with all the other features of the disease—its pathology, clinical features, and relapsing course; the periodicity in all these characters which marks the course of the disease from first to last; its onset, so sudden in many cases that patients previously in robust health can be reduced to the greatest depths of anæmia in two or three months; the no less remarkable power of recovery from it, so that patients at the brink of death can regain their health and their blood within a period of two or three months—the above feature of seasonal incidence and recurrence appears to me to be the most conclusive evidence yet afforded of the specificity of this great disease, inexplicable on any other ground than that applying to other infective diseases (e.g., scarlet fever, measles, &c.) which show similar seasonal incidence, viz., the existence of specific infection.

If any group of features connected with sex, age, pathology, clinical features, mode of onset, seasonal incidence and recurrences, and periodicity, can ever entitle an anæmia to constitute an infective disease, then the foregoing may be considered an example of such a disease in the most idiopathic form. Specificity extends through all its features from first to last, even to its sex and age-incidence—Male sex, 71 per cent. : age over 40, 85 per cent. ; whereas all other anæmias are notoriously far more common in the female sex and under the age of 40.

The disease therefore deserves the name "idiopathic" originally given to it in English medicine, and the name "hæmolytic" which, in my observation, chiefly distinguishes it—"idiopathic hæmolytic anæmia" (Hunter)—or the still more distinguishing name of "glossitic hæmolytic anæmia."

This title is the more appropriate, since it is the study of its hæmolytic and glossitic features, and the importance I have attached to them throughout that has led me step by step to recognize its full identity.

Conversely, it is the slight importance attached to such features and pigment changes by many observers that has led them and still leads them to entitle as "pernicious" cases which, in my observation, are non-hæmolytic in their pathology and essentially of the class of anæmias which I have termed "septic anæmia" in their nature and ætiology.

#### *Seasonal Incidence in Twenty-two Cases (Table II).*

These remarkable features of seasonal incidence are well brought out in twenty-two cases which I have tabulated in Table II. The cases have been chosen not on account of their incidence, but solely because they happen to be cases illustrating the character of the blood changes in different years of the disease from the first year up to the fifteenth.

It will be seen that out of the total number of instances (sixty-seven in number) in which the month of onset or relapse was ascertained, no fewer than fifty of these were in July, August and September, eleven were in October, a total of sixty-one for these four months. Two were in June and the other four were in February or April.

The twenty-two cases included five cases in which the prominent features were those of combined sclerosis of the cord. It will be seen that the incidence in onset and relapses of these cases (7, 8, 9, 10, 11) was exactly the same as in the other cases. The only difference noticeable is that on the whole they pursued a more chronic course, excepting one case (Case 7) which died nine months after onset. The other four (Cases 8, 9, 10, 11) ran a chronic course, two of them dying in three years and three months and five years; the third, being of four years' duration when last seen. The fifth patient (No. 11)

TABLE II.—INDIVIDUALITY AND IDENTITY OF THE HÆMOLYTIC DISEASE—"PERNICIOUS ANEMIA" (GLOTTIC, HÆMOLYTIC ANEMIA).  
*As shown by its Seasonal Onset and Relapses in twenty-two Cases, including five Cases of Sclerosis of Cord (Cases 7, 8, 9, 10, 11).\**

No.	First seen	Sex	Age	Onset	Second year	Third year	Fourth year	Fifth year	Sixth year	Result	Duration		
1	Feb., 1908	M.	48	July, 1907	—	—	—	—	—	Died March, 1908	8 months		
2	Aug., 1900	M.	47	April, 1900	—	—	—	—	—	Died September, 1900	8 months		
3	Jan. 1913	M.	47	Sept., 1912	Oct., 1913	—	—	—	—	Died December, 1913	1 year 4 months		
4	Sept., 1907	M.	55	Aug., 1906	Sept., 1907	—	—	—	—	Died December, 1908	2 years 5 months		
5	July, 1919	F.	34	Aug., 1918	July, 1919	July, 1908	—	—	—	Died July, 1921	3 years		
6	April, 1901	F.	41	Sept., 1899	Sept., 1900	Oct., 1901	July, 1921	—	—	Died January, 1902	2 years		
*7	Feb., 1901	M.	53	Oct., 1900	—	—	—	—	—	Died June, 1901	8 months		
*8	Sept., 1909	M.	48	Oct., 1906	July, 1907	Oct., 1908	Sept., 1909	—	Sclerositis, Aug., 1909	Died December, 1909	3 years 3 months		
*9	Mar., 1911	F.	45	Aug., 1910	(1911)	(1912)	(1913)	(1914)	Sclerositis, 1915; Sclerositis, Jan., 1901	Died 1915	5 years		
10	Aug., 1911	M.	30	Aug., 1910	Sept., 1912	Sept., 1913	(1914)	?	Sclerositis, Ap., 1913	Unknown	4+ years		
11	July, 1912	F.	48	Aug., 1910	Aug., 1911	Oct., 1912	(1913)	(1914)	Sclerositis, Dec., 1912	Recovery 1913-22	12+ years		
12	July, 1900	M.	37	Aug., 1898	Aug., 1899	July, 1900	Aug., 1901	July, 1902	Sclerositis, July, 1903	Recovery 1900-18; died 1918 of another disease	20 years		
13	Oct., 1901	M.	41	July, 1901	Sept., 1905	Feb., 1906	Feb., 1907	—	—	Died April, 1907	2 years 10 months		
14	Oct., 1911	M.	33	Aug., 1909	July, 1910	Aug., 1911	Sept., 1912	—	—	Died November, 1912	3 years 4 months		
15	Jan., 1920	M.	47	July, 1916	Sept., 1917	Sept., 1918	Sept., 1919	—	—	Well 1922	6+ years		
16	July, 1909	F.	56	Aug., 1906	July, 1907	July, 1908	July, 1909	Well	—	Died June, 1910, of another disease	3+ years		
17	June, 1909	F.	50	July, 1907	July, 1908	June, 1909	July, 1910	—	—	Died 1910	3+ years		
18	Aug., 1909	F.	39	Oct., 1908	Aug., 1909	June, 1910	—	—	—	Died December, 1910	2 years 3 months		
19	Dec., 1901	M.	43	Aug., 1900	Sept., 1901	—	—	—	—	Died April, 1902	1 year 9 months		
20	May, 1901	M.	51	July, 1900	Oct., 1901	—	—	—	—	Died November, 1901	1 year 5 months		
21	Mar., 1920	F.	58	June, 1918	July, 1919	Well (1920)	(1921)	Feb., 1922	—	Died May, 1922	4 years		
22	Nov., 1913	M.	58	Aug., 1910	Well (1911)	Oct., 1912	Oct., 1913	Aug., 1914	—	Died November, 1914	4 years 3 months		
Total cases 22					M. 14 F. 8	18		14	9	3	1	Total	67
					Incidence of attacks	Per cent.		Per cent.		Per cent.		No.	Per cent.
					July-Sept.	17		77	80	2	67	50	74
					October	4		19	1	—	—	11	16
						96		100	80	67	—	61	90

is still alive twelve years after onset of the disease, and ten years after she had cord features with ataxia. These features lessened a year after their first appearance (December, 1912), but are still present (1922) in a slowness and uncertainty in her gait, with absence of reflexes. But there has been no anæmia since 1913.

(b) *Character of Clinical Features (e.g., Glossitic and Hæmolytic).*

The identity of these features with those of pernicious anæmia (Case 12) may be illustrated by the details of the following case.

*Case 8, from a Clinical Point of View.*

Patient, a gentleman, aged 48, first seen by me in September, 1909, wildly ataxic with all the features of subacute combined sclerosis; red cells 85 per cent., colour index 1·07. By the end of two months his ataxia had lessened so much that he was able to walk and leave the home; but his blood fell to 34 per cent., with 1·5 colour index, and after a brief interval of betterment he died on December 12, 1909.

The history of his illness from first to last, when ascertained from various sources and pieced together, was the following:—

*Onset.*—He was in good health up to October, 1906 (*note period of onset*), when he had an attack of some form of *colitis* (termed “dysentery”) while in Khartoum, which laid him up for six weeks. He had never been well since. He came home early in 1907, and got rapidly well and hunted as usual. There were no nervous symptoms.

*Second Attack, July, 1907 (Seasonal Recurrence).*—He felt ill and *horribly depressed*, and was seen by his doctor. He had no physical signs except distension of stomach (*gastric features*). His teeth were very bad, suppuration very marked.

In January, 1908, when next seen, he presented a *very bad colour*, yellow, earthy, remarked on by his friends (*hæmolytic features*); and when seen by his doctor in April, 1908, he complained of *tingling* in both hands and tips of fingers, with blunting of sensibility to touch in both hands, also of feelings in his legs as if his trousers were too tight, and “the wind was blowing through them” (*peripheral nervous features*). His weight was 11 st. 12 lb.

*Third Attack, October, 1908 (note Seasonal Recurrence).*—He looked very ill, suggesting malignant disease; had lost 4 lb. in weight, felt sick and suffered from splashing in stomach (*gastric features*). Knee-jerks diminished but no loss of sensibility to touch. He continued to hunt as usual, till one day in November he fell off his horse; he got up and rode his horse for an hour or two, but afterwards said he had no recollection of the accident. He was placed in a nursing home in Dublin, where he was found to be extremely ill—profoundly anæmic (red cells 27 per cent., colour index 0·85). His mind was absolutely blank for a week, and the doctor in charge thought the prognosis was death (*toxæmic attack*). The doctor found that there had been numbness of legs for a long time (*peripheral paræsthesia*), but there was at that time no ataxia; the knee-reflexes were present, but diminished. He thought originally that the case was one of pernicious anæmia; but the improvement was so rapid that he began to doubt this (*viz.*, a rise in red cells up to 72 per cent., with colour index 0·9 in four weeks' time, and then up to 93 per cent., with colour index 1·01 two weeks later). He thought also of peripheral neuritis; also of the possibility of an arsenical neuritis, but the numbness had come on before arsenic was given.

Although at death's door in the middle of November, 1908, his recovery was so rapid that he was able to go to a Convalescent Home in Folkestone at the end of December. There he remained for about a fortnight, and then returned to Ireland, where he resumed his hunting, apparently perfectly restored to health. (Blood condition in February, 1909: Red cells 99 per cent., colour index 0·8.)

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Note of physician who attended him, November, 1908: "It is altogether a very strange case—the blood condition was so extremely grave when he first came to see me in November last (red cells 26 per cent., colour index 0·85) that the prognosis seemed death. But under treatment he improved in a marvellous way (up to 93 per cent., with colour index 1·01 in six weeks' time) that I began to doubt my original diagnosis of pernicious anæmia. The illness has had so many anomalies that a dogmatic diagnosis or prognosis seems impossible." The case here referred to was a typical case of the disease associated with nervous features, and ending up with wild ataxia and other features of subacute combined degeneration of the cord.

In March, 1909, he was not so well, and returned to Dublin chiefly with reference to some trouble with his nose—discomfort and loss of smell—trouble first noted when he was ill in November, suggesting pus in the left antrum. His blood condition had fallen (red cells 69 per cent., colour index 1·01).

#### *Septic Complications.*

Examination of the nose revealed a stream of pus flowing from the inferior turbinated body into the nasopharynx. The antrum was punctured through the inferior meatus and washed out, and on transillumination there was a marked shadow on the left side, showing thickening of lining membrane. A radical operation was recommended, and this was done on May 1, 1909. The antrum was opened up through the canine fossa, the entire lining membrane of the cavity taken away, a large opening made into the middle meatus of the nose, and any ethmoidal cells that appeared to be involved also broken down. A similar opening was made into the inferior meatus, leaving the inferior turbinal bone *in situ*.

(Note here the severe degree of antrum sepsis associated with the case, probably existing unnoticed for a long period, and accompanied by a very severe degree of oral sepsis, observed when the patient was first seen about two years before, July, 1907.)

Following the removal of the antrum sepsis on May 1, the blood condition improved, rising by May 26 to 83 per cent. of red cells, and with a fall in colour index to 0·5. Two months later (July) it was 72 per cent., with colour index 0·9, and at the end of August it stood at 84 per cent., with colour index 1·07.

#### *Sclerosis of Cord.*

*Fourth Attack, September, 1909 (note Seasonal Recurrence).*—Although the blood condition had thus improved, and had indeed remained fairly good from the date of his severe illness in November, 1908, the *nervous features had become much more marked*. When first seen at the end of August, 1909, he was wildly ataxic, scarcely able to stand, and almost falling when he turned. Knee-jerks brisk, ankle-jerks absent in left foot, brisk in right; plantars showed reflex response. There was no cutaneous anæsthesia or analgesia, but vibration sense was lost in lower limbs, normal in upper limbs. There was no muscular atrophy, and joint sense was normal in upper and lower limbs.

The cerebro-spinal fluid was not under pressure, it contained three cells per cubic centimetre, large and small mononuclear in equal number. Noguchi's test negative, ammonium chloride gave a faint haze; condition of cerebro-spinal fluid excluded tabes. For several months past his legs had felt stiff, and for the last month they had been very tottering. There were no lightning pains, no diplopia, no bladder trouble; his speech and articulation were normal, the pupils small, reacting sluggishly to light, but briskly to accommodation, cranial nerves otherwise normal, no ataxia of upper limbs (report of Dr. Purves Stewart).

As stated, his blood condition at this time (August 21, 1909) was 84 per cent. of red cells, with colour index 1·07, and the only other feature of trouble at the time was a recent recurrence of his old bowel symptoms—namely, some *diarrhæa* accompanied by slight *tenesmus*, *mucus* and a little *blood*.

*Cerebral and Mental Features.*

But the *nervous features* included some others that did not appear on the surface, but were only ascertained by me by piecing together information from various sources. They were connected with change in disposition, difficulty in management, suspicions on the part of the patient about all around him. These dated from the time of his severe anæmic illness in November, 1908, a year before, and had become much more marked since. His disposition had become altered, he had become very suspicious, very secretive about his affairs, did not like to keep an account at his local bank lest others should know about his affairs; thought that everyone was discussing his illness, and speculating as to who would take his place. When in town he used to walk behind taxicabs to escape notice. Felt as if he could not trust his brain, and had great failure of memory.

These mental traits were illustrated in a striking way on the occasion when I first saw him, early in September, 1909, in a nursing home in consultation with his doctor and a well-known neurologist, who had found the nervous features I have just described. The patient had seen his ordinary doctor under whose care he had been for some years (doctor No. 1), who found him so ill that he sent him at once to a particular nursing home. The patient instead of going there consulted a distinguished surgeon (doctor No. 2) without mentioning that he had just seen doctor No. 1. The surgeon found him so ill that he sent him at once to another home, and called in the help of a neurologist (doctor No. 3)—neither of them knowing that the patient had seen his own doctor that morning, and being under the impression that all the patient's illnesses had been in Ireland. His own doctor (doctor No. 1) then found that his patient, whom he had sent to one particular home, was under the care of two specialists in another home: and it was at this stage I was asked to see the patient in connexion with all concerned.

The nervous features then presented were those already described. He still had occasional tingling feelings in fingers of both hands, no cutaneous anæsthesia or analgesia, vibration sense lost in bones of pelvis and lower limbs, normal in spine and upper limbs. Slight clumsiness in picking up small objects, especially with right hand, gait distinctly ataxic as before. Knee-jerks normal, ankle-jerks faint in right, absent in left. Plantar reflexes, flexor response in right, indefinite in left.

The blood condition on August 22, 1909, had shown red cells 84 per cent., with colour index 1.07. The red cells showed only slight variation in size; for the most part they were large and well shaped, but there were some small ones, and a very few poikilocytes. They stained normally, and there was no evidence of degenerative changes (polychromatophilia, polychromasia); no nucleated varieties were found in several films.

At the time I saw him (September 1, 1909), his disease had become active and the blood condition had fallen to 65 per cent. red cells, colour index 1.2. The tongue was clean, but some of the teeth were carious, and all of them showed marked pyorrhœa around the roots; two or three of the carious teeth were removed three weeks later, and the degree of sepsis connected with them was such that for the next three weeks spicules of dead bone continued to come away. A dental report (by Mr. J. G. Turner) on their condition (September 28) showed pyorrhœa around all the teeth, most marked around the incisors, with tartar on the necks and in the pyorrhœa pockets.

The chief presenting features were very dark *bilious stools* and *high colour of the urine* (denoting active hæmolysis), slight *diarrhœa* with offensive stools (intestinal features shown in the urine by indicanuria), slight degree of *irregular fever* with perspiration at night.

*Subsequent Course.*

This case was under my observation for two months in a nursing home during the last four months of his illness (September to December, 1909).

For the first three weeks the chief features presented were slight *looseness of bowels* (intestinal feature) accompanied by *bilious stools* and by *high colour of the urine* (hæmolytic feature)—features denoting activity of the disease. They included,

however, another feature of essential significance in relation to the nature of the case of which no mention was found in the previous history of the case, and of which, but for my observation, no notice would probably have been taken in its later course. This feature was a recurring *sore tongue*—in his case very slight in degree.

When I first saw him (September 1, 1909) the tongue was clean and showed nothing obviously abnormal; but a fortnight later (September 13), while still looking very clean, it showed the slightest redness at the right edge near the tip and it was slightly tender on smoking; three days later this condition persisted and in addition there was a slight red patch on the right edge far back. During this period *hemolysis* was *very active*, the *stools* being *very bilious* and the *urine* of *high colour*. At the end of this period (September 18) the patient was feeling better, but his pallor had increased, and on examination it was found that his red cells had fallen from the percentage of 85 on admission into the home on August 22 down to 58 per cent. on September 17.

The attack which had thus been on him lessened from September 20. The patient felt much better in himself, the urine was not so high coloured, and by September 24 it had become paler than at any time since September 10: it showed neither urates nor indican during this period. Moreover the discharge from the nose had been got under control, and his sense of taste and smell had returned for the first time in eighteen months. Further, two or three septic teeth had been removed and an attempt made to lessen the existing sepsis and pyorrhœa present in other teeth.

By this time his resistance to septic infection had been definitely increased. On September 13 the opsonic index against a streptococcus and *Bacillus coli* isolated from stools had been found to be: Against streptococcus, 0·8; *Bacillus coli*, 0·64; and a vaccine had been given— $\frac{1}{2}$  million streptococci and 10 million *Bacillus coli*. On September 17 the opsonic index had risen to: Streptococcus, 1·11 and *Bacillus coli*, 1·01; and the vaccine was repeated. On September 28 the index against streptococcus had further risen to 1·2. But on this date, September 28, another exacerbation occurred, shown by *looseness and discomfort of bowels*, attended by higher colour of the urine with *urobilinuria*, and by *bilious stools*. The intestinal disturbance was further accompanied by another feature of special interest, to which the patient himself drew attention, namely, more tingling in his fingers (*peripheral paræsthesiæ*) such as had been present to some degree or other from the first. Between October 3 and October 11 a similar exacerbation of hemolysis occurred—*bowels loose, stools bilious, urine higher in colour*, and again he complained of *peripheral sensations* in his legs, extending up to his waist, "as if his skin had been rubbed with sandpaper."

Although the effect of these exacerbations of hemolysis had been to lower the blood count materially—namely, down to 48 per cent. red cells, with colour index 1·14, as compared with 85 per cent., with colour index 1·07, on admission on August 22, the general condition of the patient had much improved. He felt much better; his complexion was much clearer, although paler; the conditions in the nose and mouth had been much improved; and finally, by October 3, he had been able to sit up for two or three hours every morning. Most interesting of all, his ataxia was much less than it had been for some months and his gait was much steadier. He could now stand and turn about more firmly than he had been able to do.

Altogether, by October 12, he was feeling and looking so much better that he was able to go out daily for a drive, and eventually he left the home for convalescence at the seaside about a fortnight later. He improved there further for a month and then returned and attempted to resume his usual club life in London. He did all this despite the fact that when he went out his blood condition had not improved, but had actually fallen—e.g., down to 23 and 42 per cent. The last count I have (November 10) when he was convalescing showed only 36 per cent. of red cells with 1·2 of colour index—a condition which seemed hardly consistent with his feeling of betterment. But it also showed another blood change which in my experience is more important as an evidence of the degree of resistance of the blood at any time, namely, a very high polymorph count—the percentage of polymorphs at the time being no less than 183 per cent. of their normal number (5,000).

But the duration of betterment was very short. About a fortnight after he came



back he suddenly relapsed and died on December 12, 1909, after an illness dating from first to last a little over three years (since October, 1906).

(For course and blood changes see Table V, p. 36.)

(c) *Blood Changes in Typical Cases of Pernicious Anæmia, Glossitic Hæmolytic Anæmia, compared with those presented in Cases of Combined Sclerosis (Table V).*

This great feature of seasonal incidence has thus had the double result not only of establishing, as nothing else could do, the great identity of this disease (A) but also of identifying the character of the severe anæmia with which combined sclerosis may be associated. For no anæmia has such a character, least of all the form of anæmia which I have termed "septic anæmia" (B)—the only anæmia that can resemble in some of its clinical features and blood changes the above-mentioned glossitic hæmolytic disease.

I have now to add that the identity of the anæmia associated with combined sclerosis is further confirmed by its blood changes. These are shown in Table III from five cases of this condition, and compared with those presented by eight cases of most typical glossitic hæmolytic anæmia (pernicious anæmia). In an individual case (10) they are shown in Table I (p. 4), compared with blood changes in two cases of pernicious anæmia and one case of septic anæmia.

It will be seen that the correspondence between the two sets of cases is complete as regards their blood changes. It applies not only to the changes as a whole, but also to the blood changes presented in different years of the disease from the first to the sixth; to their lowest and highest and average percentages of red cells in the different years; to the character of the colour index—whether above normal, normal or below normal.

The degree of identity of the blood changes presented by the anæmia in these five cases of sclerosis with those presented in eight typical cases of pernicious anæmia is brought out in Table V. I have tabulated these in relation to the year of the disease in which they were found—viz., in the first year, two cases; in the second year, one case; in the third year, three cases; in the fourth year, three cases; and in the fifth year two cases.

In the first year the blood showed percentages as low as 15 and 20 per cent., as high as 81 per cent., average 39 per cent. (as compared with corresponding figures of 18 per cent., 85 per cent., and 50 per cent. respectively in the typical group).

In the second year the one count seen at this stage showed 63 per cent. (as compared with an average of 53 per cent. in the typical group).

In the third year the blood showed percentages varying from 27, 38 and 43 per cent. up to 99 per cent., average 61 per cent. (as compared with corresponding percentages varying from 20 up to 94, average 55, in the typical group).

In the fourth year the blood showed percentages varying from 25, 34 and 61 per cent. up to 99 per cent., average 55 per cent. (as compared with corresponding percentages varying from 26 up to 92, average 61 in the typical group).

In the fifth year the level was raised, the percentages ranging from 61 per cent. up to 98 per cent., average 82 per cent. Altogether up to the fourth year low percentages might be found: but after that year the lowest percentage was only 61 per cent.; and the average was 82 per cent.



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TABLE III.—PERCENTAGES OF RED CELLS AND COLOUR INDEX IN DIFFERENT YEARS OF THE DISEASE IN :—

(A) *Eight Typical Cases* (Cases 2, 3, 4, 5, 12, 21, 22, 23 in Table II.)

(B) *Five Cases with Combined Sclerosis* (Cases 7, 8, 9, 10, 11 in Table II.)

## (A) *Eight Typical Cases.*

	Totals	First year	Second year	Third year	Fourth year	Fifth year	Sixth year	Seventh to fifteenth year
Cases ... ..	8	4	5	4	4	3	1	1
Total counts ...	86	15	22	19	11	7	5	7
<i>Colour index:</i>	No. Per cent.							
Above normal	72 83	14	16	16	9	5	5	7
Normal ...	8 9	1	3	3	1	0	0	0
Below normal	6 7	0	3	0	1	2	0	0
		Index Red cells	Index Red cells	Index Red cells	Index Red cells	Index Red cells	Index Red cells	Index Red cells
Lowest ... ..	0·8	18 1·6	8 1·5	20 1·3	26 1·2	25 1·3	31 1·6	81 1·4
Highest ... ..	1·7	85 1·13	90 1·0	94 1·1	92 1·05	75 0·9	85 1·4	106 1·1
Average ... ..	1·16	50 1·3	53 1·15	55 1·12	61 1·15	63 1·16	58 1·4	90 1·2

## (B) *Five Cases with Sclerosis.*

							Totals B	Totals A
Cases ... ..	5	2	1	3	3	2	5	8
Total counts ...	52	7	1	18	18	8	52	86
<i>Colour index:</i>	No. Per cent.						Per cent.	Per cent.
Above normal	43 83	7	0	12	17	7	43 83	72 83
Normal ...	1 1	0	0	1	0	0	1 2	8 9
Below normal	8 15	0	1	5	1	1	8 15	6 7
Lowest ... ..	0·5	15 1·6	—	27 0·85	25 1·6	61 1·04	0·5	0·8
Highest ... ..	1·8	81 1·03	—	99 0·8	99 1·06	98 1·12	1·8	1·7
Average ... ..	1·1	39 1·27	63 0·8	61 1·14	55 1·05	82 1·08	1·1	1·16

This Table shows the remarkable correspondence between the blood changes in sclerosis (B) and those in typical cases of pernicious anæmia (A). This is notable throughout, in every year of the disease. But it is most striking as regards the colour index—namely, that in both groups of cases the colour index was above normal in 83 per cent. of all counts.

In other words, the degree of anæmia was such as would commonly be termed “secondary”—or so-called “non-pernicious type” of blood change—whereas the Table shows that the changes were exactly identical with those found in eight typical cases of the real primary or pernicious anæmia disease.

The correspondence as regards colour index is so identical as to be deemed almost uncanny. For the proportion of counts in the two groups of cases showing a colour index above normal is exactly the same, namely, 83 per cent. ;

TABLE IV.—CHARACTER OF BLOOD CHANGES IN TWO TYPES OF SEVERE ANÆMIA AT DIFFERENT STAGES.

(A) "*Pernicious Anæmia*" (*Glossitic Hæmolytic*).(B) "*Septic Anæmia*" (*Non-glossitic, Non-hæmolytic*).(C) *Severe Anæmia with Combined Sclerosis (Glossitic Hæmolytic)*.

	A			B			C		
	Red cells		Index	Red cells		Index	Red cells		Index
	14	...	1.4	14	...	0.7	15	...	1.6
	18	...	1.6	—	...	—	18	...	1.4
	19	...	1.2	—	...	—	19	...	1.4
	20	...	1.7	—	...	—	20	...	1.25
	22	...	1.4	22	...	1.0	23	...	2.2
	23	...	1.0	23	...	1.0	24	...	1.6
	25	...	1.2	—	...	—	25	...	1.0
	27	...	0.9	—	...	—	25	...	1.6
	34	...	1.4	34	...	1.0	34	...	1.5
	38	...	1.2	38	...	0.84	38	...	1.8
	39	...	1.23	—	...	—	40	...	1.5
	40	...	1.1	—	...	—	42	...	1.3
	42	...	0.9	—	...	—	43	...	1.2
	43	...	1.07	45	...	1.2	46	...	1.5
	44	...	1.13	46	...	1.0	48	...	1.5
	55	...	1.0	49	...	0.9	51	...	1.3
	56	...	1.4	50	...	1.0	52	...	1.4
	60	...	1.2	53	...	1.2	57	...	1.5
	60	...	0.6	53	...	1.2	58	...	1.2
	60	...	1.2	—	...	—	61	...	1.14
	62	...	1.35	—	...	—	61	...	1.3
	64	...	1.17	64	...	1.0	61	...	0.9
	67	...	1.0	66	...	1.1	63	...	0.8
	68	...	1.3	68	...	0.85	65	...	1.2
	74	...	1.1	73	...	0.5	67	...	1.05
	75	...	0.9	73	...	0.7	68	...	1.1
	78	...	0.8	76	...	1.0	69	...	1.23
	—	...	—	76	...	0.6	78	...	1.2
	80	...	0.12	82	...	0.6	81	...	1.03
	84	...	1.0	84	...	0.8	84	...	1.07
	88	...	0.9	87	...	0.85	85	...	1.11
	92	...	1.05	91	...	0.6	90	...	1.11
	106	...	1.1	96	...	0.4	99	...	1.06
	105	...	0.6	102	...	0.6	99	...	1.08
	—	...	—	105	...	0.4	—	...	—
Cases ... ..	8			5			5		
Blood counts ...	86			24			52		
Colour index:	Percentage			Percentage			Percentage		
Above normal ...	72	...	83	3	...	12.5	43	...	83
Normal ... ..	8	...	9	7	...	28.3	1	...	1
Below normal ...	6	...	7	14	...	59.2	8	...	15

This Table shows that almost every percentage of red cells from 14 per cent. up to 105 per cent. can be found in the two types of anæmia, A and B, and counts may be exactly similar for every degree. But, in general, the Type A differs notably from Type B as regards colour index—namely, 83 per cent. of counts having a colour index above normal in Type A, as compared with only 12 per cent. in Type B. In respect to all their features the blood changes in the anæmia with combined sclerosis correspond exactly with those of Type A, not with Type B.

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TABLE V.—THE BLOOD CHANGES IN FIVE CASES OF ANÆMIA WITH SCLEROSIS.

	Case 7			Case 8			Case 9			Case 10			Case 11			Summary of five cases					
	Date	Reds	Index	Date	Reds	Index	Date	Reds	Index	Date	Reds	Index	Date	Reds	Index	Cases	Counts	Lowest	Highest	Average	
Onset 1st year	Oct.,* 1900 3.3.01 19 1.4 3.4.01 18 1.4 18.4.01 15 1.6 Death			Sept., 1909 — — — Sore tongue Anæmia			Aug., 1910 27.1.11 20 1.25 21.3.11 52 1.15 28.4.11 67 1.05 23.5.11 81 1.03			Aug., 1909 — — — *Sore tongue Anæmia			Aug., 1909 — — — Sore tongue Anæmia			2 7 Reds Index	15	81	39	1.27	
2nd year	— — — — — — — — — — — — — — —			— — — — — — — — — — — — — — —			25.8.11 63 0.8 — — — — — — — — — — — —			— — — — — — — — — — — — — — —			— — — — — — — — — — — — — — —			1 1 — — — — — — — — — — — —					
3rd year	— —			7.11.08 27 0.85 4.12.08 57 0.52 15.12.08 64 0.9 27.12.08 93 1.01 8.2.09 99 0.8 3.4.09 69 1.1 26.5.09 80 0.5			No anæmia — — — — — — — — — — — — — — — — — —			5.2.12 43 1.2 16.2.12 50 1.3 5.3.12 69 1.2 26.3.12 77 1.0 — — — — — — — — —			1.2.12 38 1.8 28.2.12 52 1.7 27.3.12 57 1.5 4.5.12 65 1.4 15.5.12 61 1.14 28.5.12 52 1.4 13.6.12 42 1.3			3 18 Reds Index	27	99	61	1.14	
4th year	— —			*8.7.09 72 0.9 22.8.09 83 1.07 6.9.09 65 1.2 28.9.09 51 1.3 5.10.09 68 1.1 31.10.09 42 1.3 10.11.09 34 1.5 12.12.09 Death			No anæmia —			*11.4.13 61 1.3 19.5.13 61 1.06 — — — — — — — — — — — — — — —			15.7.12 46 1.5 4.10.12 25 1.6 2.11.12 40 1.5 28.11.12 78 1.05 18.12.12 69 1.23 1.1.13 78 1.2 2.2.13 90 1.15 3.3.13 99 1.06 20.5.13 92 1.1			3 18 Reds Index	25	99	55	1.05	
5th year	— —			— —			Anæmia Death — — — — — — — — — — — — — — —			23.10.13 61 1.04 — — — — — — — — — — — — — — — — — —			17.7.13 97 0.92 3.10.13 84 1.04 3.2.14 69 1.3 20.2.14 80 1.15 20.3.14 85 1.1 22.4.14 84 1.01 12.6.14 98 1.12			2 8 Reds Index	61	98	82	1.08	
6th year	— —			— —			— —			— —			— —			— —					
7th to 20th year	— —			— —			— —			— —			— —			1922 90 105 — — — — — — — — — — — — — — — — — —					

\* Date of onset of features of combined sclerosis. This table shows that the character of the blood changes and the course of cases of anæmia with combined sclerosis are exactly similar to those of typical cases of the hæmolytic disease termed pernicious anæmia.

## EIGHT TYPICAL CASES OF PERNICIOUS ANÆMIA.\*

Case 4			Case 12			Summary of eight cases				
Date	Reds	Index	Date	Reds	Index	Cases	Counts	Lowest	Highest	Average
Aug., 1906	Sore tongue		Aug., 1898	Sore tongue and anemia		4	15			
27.2.07	... 40 ...	1.4				Reds		18	85	50
6.3.07	... 45 ...	1.4	—	—	—	Index		1.6	1.13	1.3
20.3.07	... 57 ...	1.5	—	—	—					
3.4.07	... 76 ...	1.26	—	—	—					
7.5.07	... 85 ...	1.13	—	—	—					
23.9.07	... 23 ...	1.7		Sore tongue and anemia		5	22			
3.10.07	... 38 ...	1.4		Peripheral paræsthesiæ		Reds		8	90	53
3.11.07	... 50 ...	1.3	—	—	—	Index		1.5	1.0	1.15
13.12.07	... 54 ...	1.3	—	—	—					
25.1.08	... 59 ...	1.1	—	—	—					
6.3.08	... 26 ...	1.1	—	—	—					
10.6.08	... 51 ...	1.17	—	—	—					
20.7.08	... 20 ...	1.3	7.7.00	... 27 ...	1.3					
22.10.08	... 25 ...	1.6	10.8.00	... 67 ...	1.07					
23.11.08	... 44 ...	1.13	16.9.00	... 64 ...	1.25					
9.12.08	... 34 ...	1.15	8.11.00	... 80 ...	1.12	4	19			
30.12.08	Death		21.12.00	... 90 ...	1.0	Reds		20	94	55
—	—	—	18.1.01	... 94 ...	1.1	Index		1.3	1.1	1.12
—	—	—	21.3.01	... 90 ...	1.1					
—	—	—	—	—	—					
—	—	—	—	—	—					
—	—	—	17.8.01	... 63 ...	1.27					
—	—	—	27.11.01	... 62 ...	1.35					
—	—	—	24.12.01	... 64 ...	1.17	4	11			
—	—	—	22.2.02	... 92 ...	1.05	Reds		26	92	61
—	—	—	—	—	—	Index		1.2	1.05	1.15
—	—	—	—	—	—					
—	—	—	—	—	—					
—	—	—	28.7.02	... 70 ...	1.42					
—	—	—	3.11.02	... 50 ...	1.2					
—	—	—	27.4.03	... 74 ...	1.2	3	7			
—	—	—	—	—	—	Reds		25	75	63
—	—	—	—	—	—	Index		1.3	0.9	1.16
—	—	—	—	—	—					
—	—	—	18.7.03	... 85 ...	1.4					
—	—	—	9.9.03	... 45 ...	1.3	1	5			
*Marked nervous features	—	—	*23.10.03	... 31 ...	1.6	Reds		31	85	58
—	—	—	27.11.03	... 46 ...	1.3	Index		1.6	1.4	1.4
—	—	—	1.1.04	... 84 ...	—					
—	—	—	—	—	—					
—	—	—	5.7.05	... 91 ...	1.3					
—	—	—	6.7.06	... 81 ...	1.4					
—	—	—	11.3.08	... 90 ...	1.2					
—	—	—	5.5.08	... 87 ...	1.15	1	7			
—	—	—	6.2.10	... 92 ...	1.08	Reds		81	106	90
—	—	—	7.12.10	... 106 ...	1.1	Index		1.4	1.1	1.2
—	—	—	6.7.13	... 84 ...	—					
—	—	—	1918	Died of another disease						

\* Owing to considerations of space, only two of these cases are here detailed.

leaving a proportion of 16 per cent. for each group in which the colour index was either normal or subnormal. The fifty-two counts in the cases with sclerosis contained eight counts, or 15 per cent., in which the colour index was below normal, generally about 0·8 or 0·9, but in one case (Case 8) on two occasions as low as 0·5. But low colour indices are also found—as low as 0·6—in the course of typical cases of pernicious anæmia.

Associated with percentages of red cells of 57 and 80 per cent. such a colour index with such percentages of red cells would invariably be regarded as denoting a "secondary" type of anæmia.

And yet this case (8) four months later showed a colour index of 1·3, with 51 per cent. of red cells; and two months later one of 1·5, with 34 per cent. of red cells; and the patient died a month later, a typical case of pernicious anæmia—glossitic hæmolytic anæmia—as it had been from the onset three years before.

#### Table IV.

In relation to these changes, I draw attention to the following Table IV, which shows the great variability and also similarity from time to time in the character of blood changes presented by the two chief types of severe anæmia (A and B). The most constant distinction between the two lies in the character of the colour index, viz., *above normal* in 83 per cent. of eighty-six blood counts in "pernicious anæmia" (A), but only in 12 per cent. in twenty-four blood counts in "septic anæmia" (B); *below normal* in 7 per cent. (A), but in 60 per cent. in (B).

But it will be seen that for both types of anæmia the character of the colour index in individual cases could be reversed, e.g., Type A, showing 105 per cent. of red cells, with low colour index 0·6; 88 per cent., with colour index 0·9; or 78 per cent., with colour index 0·8; or 60 per cent., with colour index 0·6; or 42 and 27 per cent., with colour index 0·9. On the other hand, Type B might show 45 or 53 per cent. of red cells, with high colour index 1·2; or 66 with colour index 1·1; and in about one-fourth of cases (28 per cent.) might show a normal colour index with percentages of red cells of 22, 23, 34, 46, 50, 53, 64 and 76 per cent.

In relation to the character of the anæmia in which combined sclerosis occurs, the Table shows that the blood changes presented in five cases exactly corresponded with those of Type A (i.e., "pernicious anæmia"), and not with those of Type B ("septic anæmia"). This is specially so in regard to colour index, 83 per cent. of the fifty-two counts showing a colour index above normal, as compared with only 12 per cent. of the twenty-four counts in septic anæmia.

*Conclusion.*—Thus in respect of the blood changes at different periods from the first to the sixth year, the anæmia associated with sclerosis in these five cases was the type of anæmia termed "pernicious anæmia," i.e., the anæmia characterized by sore tongue and hæmolytic (glossitic hæmolytic). It was not that of "septic anæmia" (non-glossitic non-hæmolytic). But the most conclusive evidence as to the type of anæmia which was complicated by these features of sclerosis of the cord, is that afforded by the course it took, and, notably, by the seasonal variations it presented. This is shown in Table II (p. 28) in relation to the course taken by seven typical cases of the disease "pernicious" anæmia (glossitic and hæmolytic).

It will be seen that the seasonal times of onset (July to September), of advance (October to January), of remission and betterment (February to June), and relapses (July to September), were exactly the same in the five cases with cord symptoms as in the seventeen cases without such symptoms.

Finally, the characters of the clinical features presented by these five cases, as regards their glossitic, gastric and intestinal features, their hæmolytic features (lemon colour and high colour of the urine), fever and other toxic features, which they from time to time presented, were exactly similar to those characteristic of the disease—pernicious anæmia, glossitic hæmolytic anæmia.

The stage of the disease at which the features of sclerosis showed themselves varied. In two cases (7 and 9) this was in the first year; one died within eight months and one survived for five years.

In the other three cases (8, 10, 11) they did not appear till the fourth year of the disease. But in all cases nervous features of some kind or other—e.g., peripheral paræsthesiæ, "nerviness," neurasthenia, melancholia, "horrible depression" were presented from the first. One of these patients (Case 8) died within six months of the development of the ataxic features, in the fourth year of his disease; one patient (Case 10) had the features well marked when last seen in the fifth year of his disease, about a year and a half after they developed; and one case (11) still survives, thirteen years after onset of the disease and ten years after onset of features of sclerosis.

#### SECTION IV.—INTERPRETATION OF NERVOUS FEATURES IN THE DISEASE.

##### *Commentary on Case VII (figs. 4 and 7, pp. 11, 15).*

This case is one of extreme interest in regard to the early character and prominence of the nervous symptoms. These were variously interpreted as due first to "rheumatism," then to lead-poisoning (of which there was no history), then to "myelitis," and finally to subacute combined sclerosis.

The most interesting feature was the association of these with all the features of pernicious anæmia (viz., yellow complexion and anæmia), which ultimately became so great, e.g., 14 per cent. Next to the nervous features was the prominence of intestinal symptoms (*sore bowel*) throughout, viz., diarrhœa with occasional blood in the stools, a symptom common in most cases of pernicious anæmia in some degree or other. But its chief interest lies in the character of the infective lesions found post mortem in the jejunum, accurately represented in fig. 7. They were of a character very difficult to describe. They were not the lesions of an ordinary "septic enteritis" such as might have occurred in the later stages of an exhausting disease—especially when associated with the extreme oral sepsis presented by the case. They were unlike anything I have ever seen in the course of an experience extending to a thousand post-mortem examinations. The lesions were both small and large, the former so small that if they had existed alone they would have escaped notice; but easy to recognize when seen along with the pronounced lesions in their neighbourhood, which showed croupous patches of most varying extent as well seen in fig. 7. The whole character of the lesions was that of some infection creeping from point to point of the mucosa, and affecting specially the edges of the *valvulæ conniventes*. Peculiar as they were, their interest lies in their presence in the jejunum, a part—in my observation—not usually affected by ordinary septic enteritis, the seat of which is generally and chiefly in the lower end of the ileum and the colon. The second interest was their association with the marked features of combined sclerosis of the cord; and the third was the association of this latter with the pronounced and peculiar features of the hæmolytic disease—pernicious anæmia.

Their further interest lies in the fact that the patient happened to die when they were at their greatest intensity. Had the case been milder and prolonged for a further period of six or nine months, or for three or four years, and the patient then died, these lesions would also have passed away, or been presented in such small scattered areas as to be unrecognizable as definite lesions.

*Relation of Lesions to the Nervous Features.*—This case is the only case of subacute combined sclerosis I have ever seen in which death occurred at an early stage. It may be doubted whether I shall ever see another case of the same kind. It is possible that if watch were kept on the post-mortem findings in cases dying of subacute combined sclerosis in an early stage, indications of similar lesions in the intestine might be found.

When I recall how the tongue may present the most marked lesions (rawness, redness, fissures, vesicles, &c.) during life, and yet appear perfectly healthy and free from change after death, I think how impossible it must be to find lesions scattered over the whole area of the intestinal mucosa in patients dying in an almost bloodless condition.

It is this circumstance that in my judgment explains the apparent mystery of the infective lesions of this disease. The first tongue I ever examined from the disease was one that seemed post mortem to be perfectly normal. Had I cut a piece out of it haphazard and examined it histologically I might have found nothing abnormal. But I had seen the tongue during life a few days before; I had seen the lesions it then presented and I knew where to cut my sections, and I found the most severe lesions in the parts concerned as shown in the plates in the work referred to ("Severest Anæmias," 1910).

I regard the intestinal lesions in this case as representing the character of the intestinal lesions as produced by the hæmolytic infection underlying pernicious anæmia and responsible for the *sore-bowel* symptoms (of diarrhœa) which so often accompany the disease. "The illness began with sore tongue and the soreness seemed to extend down to the stomach and right through me to the back passage." That graphic description given by an intelligent patient accurately describes in my judgment the exact mode of spread of the hæmolytic infection in the body. Such an interpretation may seem to be too general, too vague to account for the chief features of the disease, and its persistent course; particularly for the association of this disease with pronounced nervous features, and even lesions of the cord.

But as a matter of fact these nervous features can, in my observation, be brought into direct relation with the lesions present in the mucosa of the alimentary tract. For they can be observed during life and examined histologically after death in connexion with the lesions in the tongue. During life the tenderness often associated with the lesions in the tongue is extreme—"like hell-fire extending down the throat to the stomach," as one patient described it to me. Microscopic examination reveals the cause of this tenderness—namely the extension of the lesions down to the nerves of the tongue, and the existence of a well-marked neuritis in these nerves (fig. 1).

The degree of involvement of the nerves is shown by another marked change in the tongue—namely, the extreme atrophy which the tongue undergoes, especially in its muscle (fig. 1). No such degree of atrophy occurs in the tongue in any other disease. To determine this point I examined a series of tongues from severe forms of wasting disease coming under my notice in the post-mortem room—diseases such as cancer of the stomach, chronic wasting diseases, septic diseases, &c. In none of them did I find any atrophy at all recognizable histologically.



In pernicious anæmia on the other hand, the tongue shows even during life very marked atrophy. When the tongue is put out it seems normal in size, but when withdrawn it falls into the back of the mouth as if shrunk up. On histological examination this change is marked by the most striking lesions and inflammatory changes in the epithelial covering and in the subepithelial fibrous tissue, followed by most intense atrophy of the muscle of the tongue. In one case I examined (*see plates, "Severest Anæmias," 1910*) the greater part of the muscle substance of the tongue was replaced by fat. The appearance presented in the sections, when the fat was dissolved out, was that shown on the plates, just as if the tongue consisted of open spaces covered in by a thin narrow shrunken and atrophied layer of epithelium, constituting the smooth glazed surface shown by the tongue during life.

This degree of degeneration of the muscle, it is to be noted, is not accompanied by any corresponding fibrotic change as if it were the result of preceding recurrent chronic inflammatory fibrosis. No such fibrotic changes are to be found. The atrophy has a direct toxic origin, for it can be seen most marked in the areas of the tongue where infective lesions, e.g., cracks, fissures, vesicles, may be present (*fig. 1*). But it also extends deep down into the substance of the tongue, and when severe affects the whole tongue.

Such a degree of atrophy may also be explained by changes connected with the nerves of the part, and in the muscle supplied by the nerves affected. These nerves, as I have said, can be seen to be affected in the neighbourhood of the lesions of the tongue. They show neuritic and perineuritic and degenerative changes and the muscle substance adjacent to them shows similar degenerative changes. Moreover, both these sets of changes are obviously related to the action of the hæmolytic lesion presented by the tongue, for they are most marked in the parts immediately subjacent to such lesions.

This is very well seen in the photograph I now show of the lesion found in a very typical case (*fig. 1, p. 9*). The section is through one of the small vesicles, which in certain cases are presented by the tongue. It will be seen that the epithelium over the vesicle is still intact; that a small open area represents the cavity of the vesicle filled with streptococcal organisms, and that immediately beneath this vesicle, which was the seat of the hæmolytic infection there is a surrounding area in which the muscle substance has lost all its structure and staining properties. It is pale, structureless, atrophied and degenerated, contrasting distinctly with the normal muscle of the deeper part of the tongue (*fig. 3, p. 11*).

Thus, as it would appear, the action of this hæmolytic toxin produced in these local lesions of the tongue is definitely neurotoxic and myotoxic, as shown not only by the changes it produces in the nerves themselves but also by the intense degeneration of the muscle adjacent.

If this be the case, these nervous and muscular lesions in the tongue throw a great light on the character of the nervous features presented by this disease, not only in the tongue, but in all other parts of the alimentary canal (e.g., mucosa of the stomach or intestine) which may likewise become the seats of its lesions. Similar effects will follow the action of the hæmolytic infection in the parts affected. In this way, as I interpret, may be explained the extraordinary degree of gastric atrophy which may accompany and mark this disease, a change constantly observed in chronic cases; also the degree of atrophy and thinness of the intestinal mucosa and muscularis mucosa which can often be found.

*Peripheral Nervous Features.*—But a more interesting manifestation of the

neuro-toxic action of the hæmolytic toxin of this disease is to be found in the great constancy with which this disease is accompanied by general nervous features—by peripheral paræsthesiæ (e.g., numbness, tingling, feelings of cold, inco-ordination of movements of fingers, &c.) such as are so common in the hands and feet and legs, and muscles of the calves, in this disease. These are so constant that I attach a definite diagnostic importance to their presence.

*Features of Cord Lesions.*—In other cases the nervous features are not confined to peripheral disturbances, but involve also the functions of the cord, as in the above case, viz., definite symptoms (e.g., loss of power, inco-ordination, loss of knee-jerks or increased knee-jerks, loss of vibratory sense) pointing to definite combined sclerosis in the columns of the cord. Such changes may, I interpret, be explained by the lymphatic extension of the toxin along the nerves of the parts affected up along the sheaths of the nerves to the cord itself, e.g., from the tongue, the stomach, or from the intestine.<sup>1</sup>

The clinical evidence supplied by my cases suggests that these more marked cord changes are connected specially with lesions in the intestinal mucosa such as those in the jejunum in the present case. For a feature of all the cases in which I have seen them prominent is that the chief disturbance has always been connected with the intestine, viz., diarrhœa as in Cases 7 and 8. If this be so, then the occurrence of pronounced cord changes in a case of this disease conversely suggests the existence of well-marked hæmolytic lesions in the small intestine, most probably in the jejunum.

*Central Cerebral Nervous Features.*—Lastly, in addition to the peripheral and cord lesions just considered, the disease is always characterized by functional nerve disturbances affecting the higher nervous system—the brain itself. These disturbances of a mental and psychical character are extremely common and play a great part in the general features of the patient's illness. They include, in my experience in individual cases, almost every class of mental disturbance, from mere irritability and change in disposition and temperament up to all sorts and degrees of monomania of suspicion, change in character, and even in one case acute mania. The change of disposition is most commonly evidenced by this suspicion and distrust quite foreign to the character of the patient, and it plays an important part in increasing the difficulties in treatment of certain cases. The characters which these lesser functional disturbances may take are very various in different patients and in the same patient at different times. They are not generally known, for the patients themselves seldom speak of them, but rather strive to hide their existence. When known, however, they are very interesting and instructive, as illustrated in one case in which I watched them for some ten to thirteen years, and in which the patient spoke to me freely about them (Case 12).

<sup>1</sup> As I originally described in *Trans. Inter. Cong. of Med., Lond., 1913.*

## Section of Medicine.

President—Dr. G. NEWTON PITT, O.B.E.

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### The Changes in Medicine and its Methods in the Past Forty-five Years:

#### PRESIDENT'S ADDRESS.

By G. NEWTON PITT, M.D.

THERE has been a far greater increase in our knowledge of disease during the past forty-five years, which covers the period of my own personal knowledge of medicine, than in any previous similar period of time, and it may not be without interest to indicate briefly some of the changes which have taken place both in our knowledge and in our treatment. The advance in knowledge and the consequent improvement in treatment have been due mainly to: (1) Bacteriology, which has indicated the aetiology of diseases and has led later on to the use of sera and vaccines; (2) the revolution in surgery; (3) the establishment of laboratories with microscopical and biochemical experts; (4) the much more minute attention which is now paid to physical signs and the correlation of them; (5) the use of improved apparatus for investigations; (6) experimental medicine; (7) the development of libraries and of systematic abstracts and catalogues of literature.

#### (1) BACTERIOLOGY.

The value of bacteriology has been the great outstanding feature of the period, and it is the recognition and cultivation of the various organisms which have enabled us to clear away a vast amount of speculation and to replace it by definite knowledge as to the aetiology of the various diseases. The most important discoveries were the organism of enteric by Eberth in 1880; of tubercle by Koch in 1882; of malaria by Laveran in 1881, and later of its life-history by Ross and others; of diphtheria by Klebs-Loeffler in 1883; and of syphilis by Schaudinn in 1905. Most of our differentiation of tropical diseases is dependent on similar knowledge. Vaccines prepared from the organisms have in some cases been of extreme value and the most striking is the preventive inoculation against enteric by Sir Almroth Wright, which has almost eradicated the disease.

The detection of tubercle bacilli has simplified our knowledge of the tuberculous processes, so that the disease is now treated at a much earlier stage and consequently with far better results, and the mortality of phthisis has been reduced from 1,875 to 372 per million living. We now know that everyone in town life is probably frequently infected, that every adult has a healed tuberculous focus, and that recovery from infection is the rule and not

the exception. Forty-five years ago the possibility of recovery was generally questioned and heredity was looked upon as the most important causative factor. Tuberculin as a means of treatment has quite failed to meet the expectations which were once entertained. The further diminution of tuberculosis in the future will mainly depend upon improving the physical condition and the surroundings of the individual. Undoubtedly sanatoria have been of very great value in indicating the necessary restrictions of a tuberculous patient, but the after-treatment often fails for working men, and many die within four or five years because they are almost always compelled to revert to their previous mode of life and, as semi-invalids for some two or three years, are unable to earn sufficient to enable them to rest when necessary and to maintain their nutrition.

The use of sera and vaccines now plays a very important part in treatment and their value will increase still more as our knowledge about their limitations becomes more defined. Some of the most useful sera are those for combating diphtheria, typhoid, tetanus, cerebro-spinal fever, and dysentery. The use of diphtheritic antitoxin serum has reduced the mortality in large hospitals from 40 to 7 per cent., yet, owing to its delayed use in many districts, the total mortality in the country shows no sign of diminution but rather the reverse.

The one common organism which the blood has the greatest difficulty in conquering is the streptococcus, and at present no very potent antistreptococcal serum can be prepared, and vaccines also often fail to save the patient when the blood has once become infected.

#### (2) THE REVOLUTION IN SURGERY.

The greatest change due to bacteriology has been the revolution in surgery. With the onset of bacteriological investigation it became possible to show whether a wound or instruments were sterile, and it was for the first time realized that the skin and dirty instruments were the origin of the infection of surgical wounds. Forty-five years ago, when only the pioneer operators were using the carbolic spray, surgeons were quite content if the pus in wounds was laudable—i.e., not offensive. Operating coats were invariably dirty; gloves, caps, sterile overalls and dressings were unknown, and as a consequence most wounds suppurated. One of the most valuable changes was the introduction of sterilizing apparatus. In the early part of the period the mortality of all laparotomies was so high that they were generally limited to a small incision to open an abscess or to drain an obstructed bowel, but operations in the pelvis, such as ovariectomies, were beginning to meet with success, as if peritonitis developed it was far from the diaphragm and might remain localized. One of the greatest changes in treatment took place when surgery became aseptic, so that it was possible to deal successfully with those abdominal lesions which were not amenable to medical treatment. The results of gastro-enterostomy, gastrectomy, hepatic and pelvic surgery, and the relief and removal of obstruction, especially of the large bowel, now form a very large and most successful part of the operations of modern surgery. At the present time the abdomen is possibly opened too frequently, on the assumption that the risk is negligible; but the number of cases in which the symptoms have not been relieved and even occasionally made worse is not generally appreciated as these cases are usually not published. The majority of cases of gastric and duodenal ulcers will heal under medical treatment, but there remain probably one-third which, owing to perforation,

adhesions, fibrosis, or other mechanical conditions interfering with peristalsis, can only be relieved by surgical treatment; with modern methods of diagnosis, also, the presence of an ulcer can be determined much sooner and with far greater certainty than formerly. One would, therefore, for both reasons have expected that the total number of deaths from ulceration would have been distinctly diminished. Yet the contrary is the case, for the extraordinary fact remains that, as the records at Guy's Hospital show, the total mortality from gastric and duodenal ulcers is at the present day greater than it was forty years ago, and not only so, but the number of fatal cases of peritonitis is also greater.

The number of fatal cases in quinquennial periods is shown in Table I.

TABLE I.

Years	Peri- tonitis	Gastric ulcer	Duodenal ulcer	Perityphlitis and appendicitis
1880-84 ... ..	176 ...	0 ...	0 ...	9 ...
1885-89 ... ..	192 ...	27 ...	2 ...	12 ...
1890-94 ... ..	257 ...	35 ...	14 ...	34 ...
1895-99 ... ..	250 ...	35 ...	13 ...	65 ...
1900-04 ... ..	316 ...	46 ...	17 ...	78 ...
1905-09 ... ..	376 ...	44 ...	22 ...	95 ...
1910-14 ... ..	277 ...	86 ...	45 ...	64 ...
1915-19 ... ..	285 ...	83 ...	38 ...	91 ...

During the period 1880-84 there were only nine cases of fatal peritonitis which were not due to cancer, tubercle, or abdominal operation for other than appendicular trouble; all but one were recognized as due to perityphlitis. It is therefore clear that there has been a steady increase in infective processes in the alimentary canal for the whole period, and the number of fatal cases of duodenal ulcers and of appendicitis increased ten-fold and eight-fold respectively in twenty-five years. In 1880 there were in the hospital five cases of appendicitis, one of duodenal ulcer, and six of gastric ulcer.

The development of the incidence of appendicitis is the most remarkable. In the *Transactions of the Clinical Society* there were only ten papers on the subject in the twenty-three years 1868-90, while there were sixteen in the next seven years. Examination shows that at Guy's Hospital there had only been sixteen deaths from perityphlitis or appendicitis between January, 1880, and June, 1889. In the latter half of 1889 there were five, ten years later the yearly average was thirteen, and for the past fifteen years it has been over twenty. About 1889 not twenty cases of appendicitis were admitted yearly into the hospital, ten years later there were 100, and now there are between 300 and 400. In the early days the small figures were not due to cases not being recognized, because an examination of the post-mortem reports for the three years 1880-82 shows that there was only one case of peritonitis during those three years the cause of which was not ascertained and which might possibly have been due to appendicitis. The inflammation formerly was also much less acute, as, out of eleven cases in 1888 the appendix was only removed in one, and there were no deaths. Sir Charters Symonds was the first to remove an appendix during a quiescent period; this was in 1885. During the early part of the period the cases were admitted into the medical wards and the mortality was small; as the type changed surgical interference became necessary at once and in 1911 three-quarters were admitted into the surgical wards, now all are placed in surgical wards except one or two that have not been recognized on admission. It is worthy of note that influenza started late in 1889 and has been prevalent ever since.

## (3) LABORATORY METHODS.

Another notable advance is in the information which can now be obtained from the microscopical, cultivational and chemical examinations, of the secretions, the excretions, and the blood. These have greatly extended the range and accuracy of our knowledge. The staining of sputum was not general till 1885. The staining and counting of blood cells are now so familiar that we are apt to forget that these examinations were not in use until after 1898. The Durham-Widal reaction for typhoid dates also from that time and the Wassermann reaction from 1906. The estimation of urea and of sugar in the blood is of quite recent date. Unfortunately it is generally necessary to refer these examinations to a laboratory and to trust to someone who has not seen the patient. There is a tendency on the part of a practitioner to rely upon the report to the neglect of his clinical acumen. It would appear probable that team-work in private practice will become not infrequent, but it will be essential that the final judgment on all the evidence should rest with the clinician.

## (4) ATTENTION TO PHYSICAL SIGNS.

Much more minute attention is now being paid in medical cases to the physical signs, and their number has been greatly increased.

The researches of the French school, and of Ferrier, Horsley, Head, Sherrington, Gordon Holmes, and others have revolutionized our knowledge and our mode of examining nervous cases, and this is very apparent if such books as those of Wilks or Ross on nervous diseases be compared with a modern work. The association of physical signs as syndromes, the minute attention to the changes in sensation, motion, and reflexes, and when necessary a lumbar puncture, now enable us generally to make an accurate diagnosis of the nature of a nervous lesion and its site. Although great advance has been made in diagnosis, the improvement in the results of treatment of most nervous diseases has not been appreciable, as at present the patient does not come under observation sufficiently early, and many of the nerve changes have already become irreparable.

## (5) APPARATUS FOR CLINICAL INVESTIGATION.

Sigmoidoscopes, bronchoscopes, arteriometers, electro-cardiographic apparatus, and more especially X-rays, give accurate information which was not formerly obtainable, but the last far outweighs the value of all the rest. The use of X-rays with bismuth meals, enables us to observe the movements and to map out the contour of the alimentary canal from end to end and to preserve permanent records of them. The recent introduction of more powerful machines will lead to the discovery of more minute changes and also possibly, aided by the deep injection of gas, to the definite outlining of the solid viscera. The X-ray investigation of the vascular and respiratory organs in the thorax provides one of the most valuable means we possess. X-rays and radium are also of increasing therapeutic value, but the introduction of very powerful machines will prove a source of extreme danger to their habitual users unless more stringent precautions for their daily use are enforced.

## (6) EXPERIMENTAL MEDICINE.

Much experimental work on animals has been carried out during this period. We have learnt by this means how to improve operative technique—to obtain accurate knowledge of the tracts which degenerate in the central



nervous system corresponding to definite localized lesions and to study the changes and lesions which the various organisms can produce. The removal of the thyroid by operation has led to a great increase in our knowledge of its functions and concerning the myxœdema which follows its complete removal. This line of research by experiment has very greatly advanced our knowledge and treatment of disease.

#### (7) FACILITIES FOR LEARNING.

At the present time there are greatly increased facilities for learning what is known and what has been published on various affections, by means of the *Index Medicus*, the *Centralblätter*, and various periodicals devoted to special subjects which systematically abstract all current literature. There is no doubt also that the amalgamation of the societies into the Royal Society of Medicine with its splendid library has been of great value to us all.

#### CHANGE OF TYPE OF DISEASE.

I have already alluded to the greatly increased frequency and virulence of intestinal ulcers in the stomach and duodenum. The most striking instance of change of type is appendicitis, which from being a negligible disease in 1880, when not a single case was admitted to a surgical ward and only one occasionally to a medical, remained infrequent until 1889 or 1890. Over 350 cases are now admitted each year, and it has become a daily danger, and in spite of early operation the total mortality has steadily increased. Influenza also has been a perennial scourge of recent years, and may possibly be the cause of the universality of appendicitis, as the great increase in frequency followed on the epidemic onset of influenza.

The most striking example of a diminished virulence has been in scarlet fever, the mortality of which has decreased from 675 to 38; in the "sixties" it was nearly 1,000. It was formerly a very common and virulent disease with a mortality as high as 30 per cent., this fell in 1900 to 5 per cent., and is now less than 1 per cent., so that the disease is now a mild one, as it has long been in Paris. The danger and the complications largely depend upon the condition of the adenoids and tonsils, and the mortality rapidly diminishes after the age of 5.

TABLE II.—A COMPARISON OF THE MORTALITY PER MILLION LIVING IN ENGLAND AND WALES IN 1880 AND 1920.

	1880	1920
Total mortality ... ..	19,400	12,100
Small-pox ... ..	25	0
Typhus ... ..	21	0
Typhoid ... ..	261	14
Scarlet fever ... ..	675	38
Measles ... ..	478	191
Pertussis ... ..	530	117
Phthisis ... ..	1,875	872
Nervous diseases, excluding hemiplegia ... ..	over 1,000	574
Liver and gall-bladder ... ..	278	175
Diphtheria... ..	109	150
Pneumonia ... ..	979	991
Respiratory disease, excluding pneumonia ... ..	2,700	1,156
Cancer ... ..	516	1,161
Hemiplegia ... ..	1,000	874
Nephritis ... ..	418	335
Heart disease ... ..	1,352	1,413
Pregnancy and parturition ... ..	76	105
Digestive disorders ... ..	700	646



Glanders, hydrophobia, and tetanus have become excessively rare, and there have only been 250 cases of anthrax in the last fifteen years. Improved methods of treatment have reduced the mortality of the following diseases to a negligible quantity: typhus, small-pox, glanders (the last case was in 1916), anthrax, hydrophobia (the last case was in 1911), and tetanus, now that serum is employed; while the mortality from measles, pertussis and phthisis has been greatly reduced.

A very great change has taken place in the amount of stimulants which are consumed, partly due to the alteration in the habits of the people, but still more to the appreciation by the profession of the importance of temperance. Formerly, 2 pints or more of beer or porter was part of the ordinary diet for every hospital patient, and large amounts of stimulants were usually prescribed in serious illness. Now they are only given in special cases and in great moderation, much to the advantage of the patient's health.

Now, in what direction is medicine most likely to advance profitably in the immediate future? There is no doubt Sir James Mackenzie is right in insisting that our most urgent need is a fuller knowledge of the habits and slight deviations from health which, later on, lead to disease. A thorough and prolonged study of the life-history of patients will ultimately throw light upon the ætiology of degenerative changes; of this at present we are ignorant, and when the changes have taken place medicine is no longer capable of checking their progress. The mortality from renal, vascular, and most nervous diseases, and from pneumonia and bronchitis, has not appreciably diminished during the past fifty years. We have yet to learn what are the infections which ultimately produce arterio-sclerosis. The causation of nephritis, whether tubal or interstitial, is still unknown. We are beginning dimly to realize that cirrhosis of the liver is but very indirectly associated with alcoholism. It is a degenerative change which is very commonly met with in aged domestic animals of all kinds, whether dogs, cats, horses or cattle, and the causation of which is certainly not alcohol. The death of twenty horses on an Irish estate apparently due to eating ragwort have recently been reported. The chances are 7 to 1 against a chronic drunkard dying with cirrhosis of the liver, while Sir F. Mott has shown that though there are a very large number of inebriates in asylums, cirrhosis is unknown there.

Septic infection from the gums and teeth, and to a less extent from the tonsils, is a most important factor in inducing degenerative changes, and if the State would undertake the provision of free dental treatment for the poor the efficiency of the working man might be extended several years. Chronic nasopharyngeal catarrh, which is so often neglected, is the precursor of many of the conditions of bronchitis in old people.

As in the past so in the future, the results of prevention of disease will be infinitely more efficient than those of treatment. When we can prevent the heart lesions of rheumatic fever we shall most efficiently deal with the cardiac failure of young people which we do not at present treat much more successfully than formerly. Gout, rheumatoid arthritis and other degenerative lesions may be prevented in the future, at present they cannot be cured.

## The Eradication of Glanders and Anthrax in Man and Animals.

By Major-General Sir JOHN MOORE, K.C.M.G., C.B.,  
F.R.C.V.S.

WHEN we seriously consider disease in all its aspects, and in particular the ills of animal origin to which human flesh is heir, the desirability and indeed the necessity for the close touch of medical and veterinary science and the co-operation of members of both professions in control of disease, must be accepted. Common interests which are so apparent, make the association a perfectly natural one. If success in public welfare is to be achieved, the same broad road must be traversed, and though the by-ways and collateral avenues have to be explored by the two professions respectively, the same goal is before us.

For my lecture I have selected glanders and anthrax, almost the two oldest diseases known to the veterinary profession. Though old, they are by no means threadbare, and I hope to clothe them in colours reflecting the progress and the sterling good work which at all events have been shown in veterinary science and policy.

### (I) GLANDERS.

In olden days before the light of hygiene, sanitary science, and research troubled the microbial world, this disease provided our stock terror. It was our Napoleon which scourged armies and other studs of horses in close communion. It has now been bereft of any terror or danger to man or animals, for—to continue in popular phraseology—our Napoleonic enemy has been killed by that staff college graduate mallein. In no instance can the progress of veterinary science be so clearly demonstrated than by the downfall and almost total eclipse in this country of this, at one time, serious disease. Last year only three outbreaks were recorded, this year up to date only two, and we may take it that practically it stands at the door of eradication from the British Isles. Two other factors responsible for its eradication are, first, the diminution and in some instances the total abolition of large industrial studs of horses in cities, and particularly in London and the surrounding counties where about 90 per cent. of the disease occurred; and secondly, the more complete Glanders or Farcy Order of 1907, with its extended provisions issued in 1920 for compensation, and the application of the mallein test.

Relatively glanders is easy of suppression. The real combative agent in the eradication of the disease, however, is the substance mallein. No veterinary expert would ever dream of handling an outbreak without its aid. It seems a cruel fate that the Russian veterinary surgeon, Kalning, who discovered it should have lost his life from glanders. By its agency in a diagnostic test, elimination of diseased animals at a period before danger of communication is probable, becomes a matter of supreme simplicity: we are in a strong position accordingly. To illustrate the comparative ease in handling outbreaks, the sense of security, the reduction in loss of animal life and the consequent saving to the public, and the minimum amount of inconvenience imposed on owners and others which this practical diagnostic agent carries, I

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will quote two instances in my personal experience, the one in pre-mallein days and the other of recent date under the trying and difficult circumstances of war. In August, 1892, in a battery of Royal Field Artillery (110 horses and nineteen grass mules) at Cawnpore, in India, a serious outbreak of glanders occurred. Two virulent cases were discovered in the veterinary hospital and inspection of the battery revealed four more clinical cases—not a very promising beginning. According to rule the battery was sent into isolation, which was carried out in a grove of mango trees one and a half miles distant. Over thirty animals were destroyed altogether, including those showing suspicious symptoms or unthrifty look. The outbreak covered a period of several months, and during that time the battery could take no part in combined training. Successful issue resolved itself into isolation up to and over the accepted period of incubation and strict periodical inspection of every animal for clinical signs of the disease, the unit not being declared free until four months after the last case. The whole procedure was laborious, inconvenient and vexatious. Mallein had only been discovered the previous year, but its use had not been extended to India. No more fitting example of the difference in present-day handling of the disease can be furnished than by our experience during the late Great War. I speak for the British Expeditionary Force in France and Flanders only. In that theatre approximately 750,000 horses and mules took part, and in spite of the vast movements of animals, the commingling with Allied Forces, and the occupation of enemy positions of doubtful nature, our force was kept relatively free, thanks to the use of mallein and a highly efficient veterinary service.

The procedure of control briefly was as follows: Every animal on arrival in France from overseas was subjected to the mallein test at the base remount depots. Similarly on evacuation from the front to a reception veterinary hospital on lines of communication animals were tested, and again on discharge to a remount depot after cure. Remount depots and veterinary hospitals were thus kept free. Animals were also subjected to the test on being cast and sold to the French public. In the event of a reaction to the test occurring, whether in an animal evacuated from the front or otherwise, the fact was referred by telegram to the formation concerned and all animals of the unit to which the animal belonged were tested. Reactors were destroyed. Doubtful reactors were re-tested in three weeks' time. Frequently two or more tests of a unit were required at intervals of three weeks before entire freedom was assured. In the autumn of 1915 the frequent reactions encountered in animals evacuated from the front indicated a certain menace and it was decided to test the whole force. This was done during the winter, about 300,000 animals being subjected to the test. The disease was cleared out, and even though we were again threatened on the arrival of the Portuguese Expeditionary Force in the spring of 1917, and again on the return of a division from Italy, we had no more trouble. The total numbers of animals affected (reactors) were eighty-five in 1917 and thirty-six in 1918.

At first the method of testing employed was the ordinary subcutaneous cervical, which consists of a local and a thermal reaction in affected animals. The stereotyped system of temperature registration, before and afterwards, at the ninth, twelfth and fifteenth hour under the cervical method proved an impossible burden in war where large numbers of animals are necessarily dealt with, and the usual practice of laying animals out of work for forty-eight hours, was an inconvenient feature. Therefore the system known as the intradermo-palpebral method was adopted, the advantage claimed over the

older cervical method being that it is a more suitable field test, that no temperature registration is necessary, that a reaction consisting of œdema of the lower eyelid and the profuse muco-purulent tears is more readily determined and with less doubt than by the cervical method. And it was subsequently found that animals could carry out their ordinary duty, if necessary, while under the test—a fact which removed any possible objection on the part of commanding officers of units. Personally, I have no doubt in my mind as to the superiority of the test as an eliminator of glanders. In a doubtful case at the twelfth, or even twenty-fourth hour, the subcutaneous cervical method can always be applied as a control. The cost per dose is very considerably less, representing a large saving when armies have to be considered. Over two million doses were used in France alone.

Thus has an old enemy in war been defeated. The presence of glanders in a military unit, even in war, is now looked upon as somewhat of a reproach, and if encountered it only serves to illustrate prowess in suppression by means of mallein. Its extinction in civil communities in Great Britain is within measurable distance and the Board of Agriculture with Sir Stewart Stockman and his staff have reason to be proud of their labours. Once clear, the application of the mallein test on importation of horses, mules and asses, will maintain freedom.

Now for the human side of glanders. Unfortunately a considerable number of lives have been lost from this loathsome disease, the cure of which is extremely rare. Victims have been mostly persons immediately associated with horses, and regrettable accidental infection has frequently occurred in laboratory workers. No less than seven European scientists working on the disease lost their lives during the years 1891-92. One of the discoverers of mallein died from it, and a well-known member of our College contracted it, but, after a long and terrible experience, was, I am pleased to relate, cured of the disease.

Comparative figures of incidence between men and animals are not available, but the late Mr. William Hunting in his work on glanders mentioned that:—

“During the years immediately following 1899 when the spread of glanders among horses continued and increased, deaths among men began to attract some notice. It was shown that whereas the Registrar-General returned about six or seven fatal cases per annum, a larger number were probably never recognized, but registered for burial as typhoid, influenza, rheumatic fever or blood-poisoning.”

He adds that:—

“There is very little doubt that ten times as many human deaths from glanders have occurred as have been accredited to that cause by the Registrar-General.”

At the same time it is certain that man shows a strong relative immunity.

In the light of veterinary experience the application of the mallein test to human beings should make diagnosis positive. I know of one instance where this was tried, a positive reaction resulting.

However, the eradication of the disease from horses, asses and mules will carry with it a corresponding extinction in human practice, for infection in man is by direct inoculation, though Hunting from analogy with what occurs in animals upheld the theory of infection by ingestion also.

## (II) ANTHRAX.

If the advancement of veterinary science has been accompanied by a practical disappearance of glanders from animals and man in this country, the same unfortunately cannot be said of anthrax. This disease presents a much tougher proposition, and it still provides a full field of inquiry for medical and veterinary scientists and for biological and chemical research workers, and calls for the serious attention of legislators and employers of labour. Despite our present knowledge and measures of control it continues to prevail, and the situation in respect to eradication seems somewhat hopeless and well-nigh impossible. However, it will be useful to analyse and discuss certain factors in relation to the disease, and to see if any admit of diminution or change which could be worked upon to reduce incidence both in men and animals.

Anthrax, as a disease, is much more formidable in animals than in man. The veterinarian, and particularly if he has served in eastern climes, experiences it in its intensest virulency, and it is manifested to him in a fulminant or apoplectic form, more usual in sheep, as splenic fever, invariably occurring in cattle and commonly in horses, and as an external type, less severe but common amongst horses in the East. In man the forms presented are malignant pustule and the so-called woolsorter's disease.

The bacillus of the disease (*Bacillus anthracis*) through the growth of which in the fluid tissues of the body the terrible train of symptoms is produced with such startling rapidity and the virulency of which appears to be increased by certain repellent characteristics of its capsule, is easily destroyed by physical and chemical agencies, and in unopened carcasses under putrefactive changes it has a relatively short existence. But unfortunately for control, the bacillus, under aërobic conditions, forms a spore, and it is to this circumstance that all our difficulties are attributed. It is the most resistant of all known spores, and it is capable of retaining its vitality for a large number of years. Unless means are available of encompassing its death or of preventing the accomplishment of its life history, eradication of the disease is beyond the reach of possibility. All combative knowledge and energy must be directed towards it specially as our arch enemy, and necessity more particularly arises from the fact that so many animal products, e.g., wool, hair, hides, bone manure, and feeding materials for animals, are imported, each offering possible media of infection.

In studying the causal agent of anthrax not only in regard to pathogenicity, but also in regard to its life history altogether, and to its amenability to destruction, we must constantly bear in mind its analogy with other and higher forms of plant life. Conditions favourable for growth are more or less similar, and what prevents or destroys germination in the one may do so in the other. I am not satisfied in my mind as to whether the organism of anthrax in nature is a pure saprophyte, that is to say, living on dead or decaying matter in the soil, or whether it is parasitic to living plants in addition. In India outbreaks usually follow rain, chiefly in the monsoon season, and the medium of infection lies in the fresh grass supply. The clean grass supply of Government grass farms has tolled the knell of the disease so far as army and cantonment animals of India are concerned, but in the old days of unit grass-cutters, who were sent into country districts to collect grass from roadsides or any other apparently favourable spot, the disease was much more prevalent. Soil infection in India is the cardinal point of incidence, and it

may be interesting and instructive to relate examples of the longevity of the organism in soil or places. Whilst I was Senior Veterinary Officer in the Meerut Division in 1906 I took the precaution of recording small localities or pieces of ground which were proved to be anthrax-infected. The Viceroy's bodyguard at Dehra Dun could never make use of grass in the immediate neighbourhood of their sick lines, because at one time or other a post-mortem examination had been made there and blood spilt. On two occasions within my personal knowledge anthrax supervened when attempt was made to use the grass. Similarly the bodyguard horses would contract occasional anthrax from grass obtained from the halting places of the old Saharanpur-Dehra Dun Mail Tonga route in use before the railway was built, so much so that they were placed out of bounds. One can readily picture mortality from unrecognized anthrax among Tonga and Ekka ponies along that route and the consequent infection of soil. The Artillery cricket ground at Meerut was infected through the death of two bullocks, a post-mortem having been held and the buckets washed at the well in the corner of the ground. I should explain that it happened before knowledge of sporulation and of the extreme danger of spilling blood became so precise that it was subsequently drilled into everyone. A considerable number of years after a hay crop from the ground was tried, and, anthrax resulting, the stack was destroyed and a ban put on the ground. Again at Meerut during the rainy season of 1906 I was asked by a young civil servant to see his valuable pony which had died in great distress and agony during the night. The evidence of the veterinary assistant and the external appearances indicated anthrax, and a careful post-mortem examination at one of the incinerators built for the disposal of carcasses in cantonments confirmed the diagnosis. I asked the owner where his grass supply came from for the past four days, and he showed me the exact spots in his own compound. It seemed impossible to connect them with infection, but just at the moment of discussion the collector of the district joined us, and I explained the situation to him, whereupon he exclaimed: "Oh, but I remember when I joined the Civil Service, twenty-five years ago, a road passed through this compound, and there were sweepers' houses just over there" (indicating the position). The source of infection was thus immediately solved—mihitars' habitations—anthrax or doubtful flesh or skins brought to them being responsible.

There is no doubt as to the considerable soil infection existing in India and countries contiguous to it—Afghanistan, Baluchistan, Persia, Tibet. Past history and present reports point to this. I am bound to give prominence in my remarks to soil infection in countries from which animal products and foodstuffs for animals are imported into Great Britain, because it unquestionably reflects the incidence of the disease at home both in man and animals.

Sir Stewart Stockman, in his annual reports issued from the Board of Agriculture, is distinctly of opinion that soil infection at home is not so prominent a factor, but that incidence in animals is from extraneous sources. In an analysis of 1,597 confirmed cases for the years 1905-10 he shows that (a) 13·6 per cent. were traceable to a previous case; (b) in 69·5 per cent. evidence pointed to infection from artificial feeding stuffs or manure; (c) in 19·1 per cent. there was no history of former outbreaks, nor of artificial foodstuffs, nor manure. Of 197 outbreaks on previously clean farms in 1919, inquiries pointed to the probable source of origin being the use of imported feeding stuffs in 107 instances, while, in 1920, out of 399 outbreaks on previously clean



farms, 294 were attributed to imported feeding stuffs and thirty-one to both imported feeding stuffs and artificial manures. Moreover, he shows that incidence in animals is not so apparent in the season of summer when grazing is usual, but it is greater during the winter months, when stall feeding on cake, meal, &c., is in process—a very significant fact, proving Sir Stewart Stockman's view.

Industrial anthrax in human beings is chiefly from extraneous sources. In the case of wool, Persian (including Baghdad and Bussorah wools) and East Indian materials are the most dangerous, and it may be noted that the so-called East Indian wool includes, Indian, Afghanistan, Baluchistan, Waziristan, the North-West Frontiers, Tibet, and a certain amount of Persian wool sent by sea to Karachi. Of mohair, Turkey and Van skin mohair, and Cape mohair are all offenders. Skin alpaca from Peru, East Indian goat hair (other than mohair), East Indian skin Cashmere, and Egyptian raw wool also show a high degree of infection. The greatest amount of industrial anthrax in Great Britain exists in the woollen industry, and skin wool is more dangerous than shorn fleeces. Hides and goats' skins received from various countries, chiefly from China and India, and horse hair from Russia, China and Siberia, all add their quota.

From what I have said it is quite obvious that the whole anthrax question resolves itself into a world problem, and it is equally apparent that incidence and its reduction in human beings is contingent on the completeness, more or less, of the control of the disease in animals. In other words, it is primarily an agricultural, or more particularly a veterinary question, and in the framing of measures for the eradication of the disease this most essential and basic fact must not be lost sight of. I have read very carefully the *Proceedings* of the Departmental Committee on Anthrax, 1918, a most valuable and instructive publication; and I cannot but admire the care with which investigations were conducted, and agree in the main with the conclusions arrived at and recommendations put forward. But if I had to criticize the Committee's findings, in the full knowledge which I possess professionally, I should say that they should have made a stronger onslaught on the cause and gone more to the root of the evil, while allowing their excellent recommendations to stand. It is to be hoped that the Advisory Committee appointed by the International Labour Organization to report in 1923, will make the veterinary aspect the first and chief plank in their deliberations. As nearly all the incidence in human beings and 70 per cent. in our home flocks and herds is traceable to extraneous sources, it is for exporting countries of animal products and feeding stuffs, in the first instance, to initiate and maintain a plan of campaign by means of which disease in their flocks and herds can be reduced. I quite realize the difficulty and to all intents and purposes the non-feasibility of this in foreign countries in which civilization is crude and where the state of society is such that men are laws unto themselves; but even in these there are ways and means that might be tried, e.g., propaganda, classification of wool and hair which might ensure better exclusion of doubtful material, the appointment of commercial attachés to embassies or consulates who could watch over and act in home interests.

There is, however, no excuse for our Dependency of India from which so much of our raw materials of wool, hair, hides, linseed and other feeding-stuffs are derived. The amount of contagious animal disease in civil districts in India is appalling, and the means for control are absolutely inadequate. This reflects seriously on a portion of our Empire.



But what can be expected of a department—the Civil Veterinary Department—to which no head officer is permitted to co-ordinate the efforts of the veterinary staff of the department in the provinces, which numbers only thirty-five British veterinary officers, including professors at colleges, laboratories, and officers on furlough, for the whole of India and Burma, and includes an altogether inadequate subordinate staff of Indian veterinary inspectors and veterinary assistants, the reporting of disease to whom is left to the village "patwali" or revenue tax collector. There is no Contagious Diseases of Animals Act such as exists in other well-ordered parts of our Empire. A Glanders and Farcy Act has existed for many years and surra has been scheduled under it. Recently also a Dourine Act was brought into force. During my tenure of office as D.V.S. of the Army in India I pressed for a proper Diseases of Animals Act (India) in consequence of serious inconvenience and loss amongst army transport bullocks and slaughter animals of the Afghan and Frontier Expeditions of 1919, and at the request of the honourable member in the Revenue and Agricultural Department of the Government of India, brought out such an Act which was very ably edited by one of my administrative veterinary officers. It was not the Army's business as you will readily observe, but as I have previously remarked there is no directing veterinary head to the Civil Veterinary Department. The Act was most simple, and was framed in such a manner that the susceptibilities of no Indian race would be offended. It was referred to the Provinces for opinion, and I hear that after a lapse of two years nothing has been done about it, that the time is not considered ripe for its adoption, or that the usual spectre of finance precludes its becoming law.

I have discussed this factor at some length, but I am convinced that it cuts at the very root of the incidence of anthrax in our own country both in man and animals. It is quite reasonable to expect that India, in these enlightened days, should follow the example of Great Britain, Ireland, Australia, New Zealand, Canada and South Africa.

In the actual handling of an anthrax situation in animals (as defined under the Anthrax Order of 1910) there are three main and indispensable factors to be observed: (a) Compulsory notification (such notification to be extended to sudden death without apparent reason); (b) compulsory disposal of carcasses in accordance with defined procedure and prohibition of cutting up of carcasses; (c) disinfection of surroundings.

The slaughtering of sick animals on farms and private slaughter-houses and the thoughtless and illicit disposal of carcasses should be severely dealt with by law. And in the disinfection of ground it is a wise precaution (indeed, I consider it essential) to repeat the process—in other words, to practise what may be termed a fractional disinfection lest under the heat and moisture of the first disinfection a certain number of spores may have escaped destruction and have germinated.

The necessity for proper disinfection of raw material of wool, hair, hides and skins as a safeguard against human infection must be supported; but the disinfection should be carried out in the exporting country, the various international countries agreeing on this point to avoid dislocation of trade.

Effluents from tanneries and from horsehair and wool factories should be sterilized before entering a drainage system, and waste products and sweepings, generally, from these places should be prohibited from use as manure.

It is of course impossible satisfactorily to sterilize feeding stuffs for animals without destroying their properties. Linseed cake, which has so often been

blamed for anthrax, if properly manufactured, ought to be reasonably safe as it is brought to a temperature of 212° F. in manufacture. Cold pressed cakes should be discarded or used with caution. In any case proprietary or manufactured foods should be sold under a guarantee of purity and obtained from firms of repute. Indeed, the same may be applied to all imported food stuffs. However, it is certain that imported grains often owe their infection to ships having either previously, or at the same time, carried cargoes of hides, hair, wool and other animal products, and better attention should be paid to the cleansing of cargo vessels and to the safe disposal of waste material and sweepings.

Inoculation by vaccines for the prevention of the disease in animals is of little practical good, for immunity thus conferred only lasts about one year.

## Section of Medicine.

President—Dr. G. NEWTON PITT, O.B.E.

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### Experiences with the Everest Expedition.

By T. G. LONGSTAFF, M.D.

(ABSTRACT.)

ONE of the greatest difficulties at these high altitudes is that of obtaining water, as above 25,000 ft. the snow evaporates and there is no ice. The loss of body fluids by evaporation is a grave element in mountain sickness, and thirst is a severe trial at high altitudes.

The members of the Expedition were divided into two parties, one of which used oxygen while the other did not. The base camp was at an altitude of 16,500 ft., the second camp was at 19,500 ft., the third at 21,000 ft. and the fourth at 23,000 ft. The first party not using oxygen climbed from 21,000 to 23,000 ft. in four hours, that is at the rate of 500 ft. per hour, whilst the second party using oxygen did the same amount of climbing in three hours; that is, at the rate of 666 ft. per hour. In 1907, without oxygen, I climbed another peak at 600 ft. per hour. The coolies, each carrying 20 to 30 lb. in weight, also climbed to the fifth camp at a height of 25,000 ft. The first party climbed to 27,000 ft. They climbed from 23,000 ft. at the rate of 400 ft. per hour, while the second party, using oxygen, climbed the same distance at the rate of 666 ft. per hour. With the use of oxygen the rate of climbing did not vary but the party who did not use it climbed only 318 ft. per hour at the higher altitudes. Those employing the oxygen apparatus should be trained and skilled in its use.

All members of the climbing parties suffered from frost bite, those using oxygen only superficially, but the party who did not use it suffered very severely. Frost bite being literally due to deprivation of oxygen owing to the cessation of blood circulation, the deficiency of the air breathed at high altitudes produces a very "vicious circle" which retarded recovery even at the base camp.

The amount of oxygen to be taken by climbers varies, but it should be used in the smallest quantities possible at a time, so that its loss will not be felt upon its cessation. For getting exhausted men down it should be invaluable, for in their case speed is safety. Oxygen is a specific in the prevention or arrest of frost bite.

If the party could have remained long enough at the very high altitudes to get thoroughly acclimatized, they would have been able to reach the top of Mount Everest, given of course favourable weather. But a succession of accidents, either frost bite or failure of the oxygen apparatus or bad weather, handicapped them when the chances of success seemed good.

## DISCUSSION ON MEDICAL ASPECTS OF LIFE AT HIGH ALTITUDES.

Mr. J. BARCROFT, C.B.E., F.R.S.,

briefly described some of the observations made during the expedition to the Peruvian Andes (chiefly at Cerro de Pasco). This expedition was undertaken for the purpose of observing the effect of high altitude on the physiological processes of the human body. Mr. Barcroft said that certain symptoms due to high altitude—vomiting, headache, defective vision, and defective hearing—passed off after a time, but that certain alterations in appetite, temper, &c., remained. All these symptoms, he considered, were a response on the part of the body, and more especially of the brain, to a change in the nature of the blood. The members of the Expedition had examined the nature of the blood in themselves, in the resident Anglo-Saxon miners, and in the natives whose ancestors had lived at Cerro for generations, and who were therefore thoroughly acclimatized.

*Arterial Oxygen Saturation*—The speaker gave tables illustrating the saturation of the blood with oxygen. That of the members of the Expedition was 95 per cent. saturated at sea level, but at Cerro (14,200 ft.) it was approximately 80 to 90 per cent. This corresponded roughly with the oxygen saturation of the blood of the natives of Cerro. The arterial blood as withdrawn from the radial artery with a syringe was dark and of a venous appearance, yet owing to the excess of hæmoglobin present there was actually more oxygen present in 1 c.mm. of blood than at sea-level.

*The number of red corpuscles* in 1 c.mm. of blood of the members of the Expedition was about 6·5 to 7·5 millions. In the natives it was slightly higher. The nature of these corpuscles had been studied by Dr. H. S. Forbes, another member of the Andes Expedition. Certain cells show a reticulum with cresol-blue staining and are considered to be red blood corpuscles. These reticulated red cells showed a marked increase during the ascent. After the descent these cells fell to below their normal percentage. In the natives the ratio of reticulated to unreticulated red cells was not greatly increased, but the absolute number of reticulated cells per c.mm. was about 50 per cent. greater than normal. This (the speaker argued) suggested hypertrophy of the bone marrow. There were no nucleated red cells.

*"Oxygen Dissociation Curve."*—There was a definite change in the affinity of hæmoglobin for oxygen. This was shown by the rise in the oxygen dissociation curve, so that at any oxygen pressure the hæmoglobin would take up more oxygen than before.

In conclusion, Mr. Barcroft said that he agreed with Dr. Longstaff that the members of the Everest Expedition could have reached the top if only they could have stayed long enough at the different camps to get thoroughly acclimatized to the high altitudes.

Wing-Commander MARTIN FLACK

stated that flying and living at high altitudes presented different conditions and problems. In flying there was little or no question of acclimatization, since an officer who flew 240 hours in one year was in a rarer atmosphere for less than ten days in that time, allowing for time of ascent and descent.

In flying the chief immediate effects noticeable as the result of high altitudes were:—

(a) *Hyperpnoea* with increasing altitude, in some cases pronounced dyspnoea.

(b) Increase of pulse-rate with rise of blood-pressure, the latter as in muscular exercise gradually falling as the flight became prolonged. In certain cases the blood-pressure fell quickly—a marked hypotension ensuing. Cases of syncope in the air directly due to diminished oxygen tension were rare.

(c) *Impairment of Judgment*.—At high altitudes all psychomotor responses were increased in time. Many pilots complained of somnolence. Feebleness of judgment, loss of flying skill and impairment of morale were also observed.

(d) *Muscular Weakness*.—At great heights many flying officers experienced difficulty in performing muscular work, e.g., swinging machine-guns, working camera, &c.

Vertigo and vomiting were comparatively rare.

As regards remote effects, prolonged high flying brought about a general fatigue. In connexion with the respiratory system there was a marked diminution of the vital capacity, due mainly to loss of expiratory tone, which led to insufficient lung ventilation.

Certain cases developed signs of circulatory insufficiency (D.A.H.), low diastolic pressure, &c., and passed to the cardiologist; others developed more marked nervous symptoms (anxiety-neurosis, &c.) and passed to the psychologist and neurologist.

The use of oxygen at high altitudes was a necessity and did much to mitigate the ill-effects. Special attention was also directed to the clothing of the aviator. Finally, physical exercise was looked upon in the Royal Air Force as part of an officer's duty in that it rendered him better able to withstand the effects of prolonged work at high altitudes.

#### Mr. T. H. SOMERVELL

(a member of the Everest Expedition) said that he and another member, Mr. Finch, had tested their efficiency in Dr. Dreyer's "Box" at Oxford. Mr. Finch was then less affected by the rarefied air than Mr. Somervell, but on the actual expedition he (Mr. Somervell) seemed to be more fit when at heights of 20,000 ft. and over, no doubt because of his better acclimatization, a factor of supreme practical importance of which the "Box" experiment could not take account. He and several others of the party experienced occasional Cheyne-Stokes breathing when lying down at 16,000 ft., but after a few days these attacks passed off, not to recur even at much greater heights. Finch experienced mountain sickness at 17,000 ft., but not subsequently at greater elevations. Crawford was sick at 19,500 ft., but afterwards reached 23,000 ft. without ill-effects. As one went higher and higher, breathing became automatically more rapid and more deep, but in most cases no distress was occasioned. Colour-index of blood from several of the party reached 124, but a higher figure than this was never obtained. While in the neighbourhood of 26,000 ft., he (Mr. Somervell) noticed his respirations were 55 to the minute, and it was impossible to take more than one step upwards to five complete respirations, though no distress was experienced while this rate of progress was (automatically) adhered to; at the same elevation his pulse while moving upward was 180; while at rest at 25,000 ft., his pulse was about 90 in the evening, and 82 the following morning. Several of the high-climbing

party had enlarged hearts for some days after their attempts on the mountain, though in one or two cases there was no visible after-effect of this continued exertion.

To sum up, the Everest Expedition was unable to get many accurate scientific observations or figures, but it did prove in the most practical way that the human body could acclimatize at greater heights than had hitherto been attained, and could stand fairly well, with or without oxygen apparatus, a height of 27,000 ft.—and probably much higher.

Professor J. C. MEAKINS, M.D.

(a member of the Andes Expedition) said that the cyanosis of persons living at high altitudes resembled that of pneumonia. He had noticed among the natives of Cerro the frequent presence of club fingers, not associated with pulmonary or cardiac disease. Their experiments showed that the basal metabolism of some members of the expedition increased (these members were all found to have lost weight) while it decreased in others. The diffusion coefficients were lower in those members who were susceptible to mountain sickness. Measurements of the chests of the natives, when they were compared with those of the Anglo-Saxon group, showed an increase in the width of the chest relatively to the height of the person. Roughly speaking, a native of 5 ft. 3 in. in height had the chest of a man of 6 ft. X-ray photographs of the chest showed a distinct difference in the angle of the ribs; there was a greater rib slope in the case of Europeans. Radiograms were taken of the hearts of five members of the expedition at sea level either before or a month after sojourn at a high altitude; at Oroya (12,178 ft.) within two hours after arrival, and at Cerro de Pasco (14,208 ft.) after two or three weeks' residence on the sierra. It was found that the heart shadow was definitely smaller in size in those who stood the altitude well.

Flight-Lieutenant W. B. FARRINGTON, D.S.O.

(of the Royal Air Force), who had been engaged for ten months in high flying duties as a pilot in France, 1917 to 1918, spoke, giving his personal experience as to the effect of flying at high altitudes, i.e., 18,000 ft. to 21,000 ft. He said that he experienced oxygen want at 18,000 ft. and above. On first reaching this height there would be a feeling of distress—panting for breath, but after a quarter of an hour this would pass, and the remainder of the flight of two or three hours could be completed without any recurrence of the feeling.

Muscular weakness was very apparent at height. Considerable effort was needed to pick up a glove from the floor of the machine. Observers used to find taking photographs or firing their machine-guns very exhausting.

His experience was that after about an hour's high flying, the brain seemed to become tired, it became hard to concentrate on mental work requiring close attention such as difficult map reading. In this connexion also, being probably due to the same causes, there was a tendency for pilots to make bad landings after a prolonged flight at height.

After landing back on the aerodrome he invariably had a headache, but this could be dispelled by an hour or two of sleep. Some pilots took oxygen after landing, which was found effective.

With regard to withstanding the effects of high flying, that is accli-

matization, personally he had noticed neither improvement nor deterioration in this respect.

He considered oxygen taken while high flying of the greatest value, as it dispelled all the evil effects previously mentioned. With oxygen, flying at high altitudes was no more exhausting than low flying. Oxygen was taken during flight either continuously through a mask or intermittently through a cigarette holder. He always used the former method, but the latter was a less cumbersome apparatus and therefore more popular.

MR. MALCOLM L. HEPBURN

briefly reviewed the scientific data in connexion with physiological effect of high altitudes, covering a period of the last twenty years, and contrasted the opinions then formed with those of the present day.

From a mass of physiological literature the following facts emerged: (1) That the symptoms of caisson disease must be due to mechanical causes due to reduction of pressure; (2) that the symptoms of mountaineers and aeronauts must be due to the diminished supply of oxygen; (3) that acclimatization was very often confused with training, and that much of the improvement in the symptoms of mountaineers was simply due to the result of proper training.

He (Mr. Hepburn) considered that acclimatization must be only considered in its relationship to the oxygen-carrying power of the blood as applied to ordinary respiration and muscular work. He also emphasized the fact that in one way or another the appearance of symptoms and the physiological effect of reduction of pressure might be postponed on the mountains to quite considerable altitudes, and that the altitude limit of each individual must vary enormously according to his physiological capacity.

He expressed the following opinions as stated by him twenty years ago:—

- (1) Illness due to the effect of high altitudes need not be considered as a clinical entity below 17,000 ft. at least.
- (2) Fatigue must be eliminated as far as possible by careful training and ingestion of proper and suitable food.
- (3) In addition to training, high mountain climbing must be made as easy as the conditions of the mountain would permit, and that the rate of climbing must be as slow as possible in order to economize muscular work.
- (4) That a supply of oxygen would be found necessary above a certain height, which varied with the degree of acclimatization.

Therefore, if the proper organization could be carried through, if the weather would allow of the establishment of a sufficient number of camps; if the mountain to be attacked were easy, and the carrying of oxygen were rendered feasible, the physiological effects of high altitudes would not prevent the highest point on the earth's surface from being ascended by man.

Mr. Hepburn then contrasted these opinions with the position at the present time. Much valuable scientific work by Leonard Hill and others during the last twenty years had established the value of oxygen inhalation both in diminished atmospheric pressure and in the performance of muscular work; while the practical work on the mountains by properly equipped and physiologically sound mountaineers had shown the possibility of ascending to heights of 27,000 ft. both with and without oxygen. He thought that there need be no controversy about the utility of oxygen, which in any case must render the ascent of high mountains much easier, and was therefore highly desirable if the transport difficulty could be overcome.



Professor J. S. HALDANE, F.R.S.

said he did not think that Mr. Barcroft's results had shaken the evidence furnished by the Pike's Peak Expedition, that increased oxygen secretory activity of the lung epithelium was a main factor in acclimatization. And the fact that the Everest Expedition had reached a height of 27,000 ft. without the use of oxygen had considerably strengthened that evidence. In 1919 Meakins, Priestley, and the speaker had pointed out that the diminishing oxygen saturation of the blood from an artery as altitude increased must be out of all proportion to the diminution in the mean oxygen pressure in the blood leaving the lung alveoli. It was this latter value only which was concerned in the question of oxygen secretion, and that they had measured on Pike's Peak. They already knew it was useless to measure only the percentage saturation with oxygen of blood from an artery.

## Section of Medicine.

President—Dr. G. NEWTON PITT, O.B.E.

### Osteo-arthritis of the Spine.

By CLAUDE GOULDESBOUGH, M.B.

IT must become apparent to any radiologist of even a limited experience in any big hospital how very frequently osteo-arthritis is seen in the spines and pelves of cases which are sent down to his department. He will also be struck with the great diversity of the clinical reasons which have brought the patient under his notice. The first thing that drew my attention to anything beyond these facts occurred some ten years ago, when I could not help noticing how many cases sent down with a provisional diagnosis of renal calculus showed no evidence of calculus. (I believe that even the most eminent medical men are not always quite certain as to the positive existence of a calculus, or, at any rate, not on clinical examination alone.)

I, therefore, collected 196 cases taken from both the wards and out-patient department of St. Thomas's Hospital in 1913, all of these cases being sent to me with a provisional diagnosis of renal calculus, and as far as I was able, I classified them according to the only means I had at my disposal, as follows:—

	Cases	Percentage
(1) Calculi present ... ..	19	9.7
(2) Calculi not present ... ..	144	73.4
(3) Cases not re-examined ... ..	12	6.1
(4) Osteo-arthritis of spine ... ..	17	8.7
(5) Nephroptosis ... ..	1	0.5
(6) Calcareous deposits ... ..	2	1.0
(7) Bone growth ... ..	1	0.5

In view of the more perfect technique which we have now in comparison to that of 1913, it is very highly probable that the list of "calculi not present" would have been better resolved. For instance, it was then rather an achievement to show the outline of the kidneys, and so to be able to diagnose, say, a nephroptosis, with certainty. However, the main point is, that out of those cases there were nearly as many osteo-arthritic spines as there were cases of stone.

In answer to the possible objection that we missed a large number of semi-transparent stones in those days, the reply would be that we also failed to diagnose a certain number of early osteo-arthritis cases—almost certainly equal to, and probably more than the number of renal calculi. The reason for this I will explain later, but it has to do with a certain important improvement in technique. Since that date I have noticed that the diagnosis of osteo-arthritis which has proved correct has become increasingly frequent.

[April 24, 1923.]

Originally hardly any cases of early disease were sent down with that diagnosis.

The second point is that if osteo-arthritis of the spine be compared with the same disease in other portions of the body, the character of the pathological process seen in the skiagrams appears different. In the knee- and hip-joint, for instance, although the "spiky" proliferations of the auricular margins tend to curve upwards or downwards, they never apparently fuse, as they do in the case of the spine, to form a complete bridge.



FIG. 1.—Antero-posterior photograph of mid-dorsal region of dried spine showing marked fibrositis ossificans ("osteo-arthritis") of Type A.

The third point is that there seem to be three types of osteo-arthritis of the spine—two completely distinct from each other, and the third a combination of the other two.

The first type (Type A, *see* figs. 1 and 2) is much the commonest, and the progress of the disease may be described as follows: The earliest indication is the formation of a small spike on the lateral borders of the articular margins of the vertebræ. These increase in size, and after a time the spike

tends to bend upwards in the case of the inferior margin (say of the third lumbar), and downwards in the case of the inferior margin (of the second lumbar), so that eventually these meet and form a complete buttress or bridge between the two vertebræ. This condition may be unilateral or bilateral, and may involve one segment, or the whole, or a considerable portion of the spine. If a considerable portion is involved bilaterally we have the "poker-back" spine, the appearance of which is as if plaster of Paris

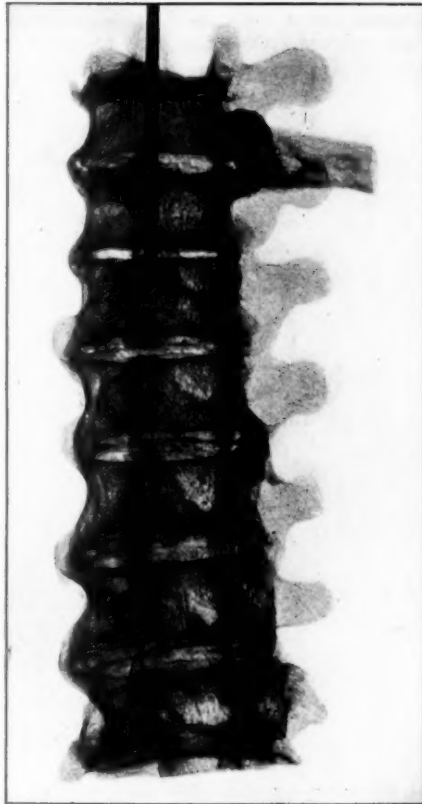


FIG. 2.—X-ray skiagram of same specimen as fig. 1.

had been poured over the spine and allowed to set. As regards the distribution of this type, so far as I can judge, the region most commonly affected is the dorsal, then next most frequently affected are the lumbar and sacroiliac regions, and the least commonly attacked is the cervical region. But it is difficult to be certain about this, because in the dorsal region the spine is considerably hidden by the heart and great vessels and early indications of the disease in this region are difficult to make out.

Recent improvements in technique have enabled us to inspect the dorsal region more clearly, and have strengthened my view on this point. Examination of dried skeletons at the Royal College of Surgeons furnish the support on which I most rely in making this statement.

□ The second type (Type B, *see fig. 3*) is uncommon, but undoubtedly it occurs. Here we have no extraneous deposits, no "spikiness," no fusion, but simply an erosion—a pressure erosion, I believe—on the fibro-cartilaginous disc between the segments, with the result that the long segments approximate till they may fuse. In the illustration of the case I am showing this afternoon this condition is fairly well marked. Objection may be raised that this is a case



FIG. 3.—(X-ray) skiagram of lumbar region in a patient illustrating Type B of fibrositis ossificans ("oste-arthritis").

of caries. If so, neither the history nor the X-ray appearances confirm this view. The patient, a man of about 48, complained of several years' history of recurrent pain in the small of the back, varying in intensity, and sometimes disappearing altogether, otherwise he was perfectly healthy. From the X-ray point of view there is no irregularity of outline, no local erosion, nor any new bone-formation.

The third type (Type C, *see fig. 4*) is a combination of the other two, and although (supposing my idea of the local causation of this disease to be correct) I have been searching for an example of this, it is only comparatively recently that I have come across an undoubted example of it, which I now show.

Here we have the erosion of the discs affecting most of the lumbar region with a distinct, but not marked attempt at buttress formation.

Now as regards the X-ray pathology of Type A. With the kind permission and help of Professor Shattock, I examined a number of dried osteo-arthritis spines (of Early Christian Martyrs as a matter of fact, from the Catacombs of Rome) at the Royal College of Surgeons. I also took ordinary photographs of them. Those now shown consist of views of the whole spine, and also of spines split longitudinally. Two interesting points arise out of these skiagrams and photographs. The *first* is that even in the dried skeleton



FIG. 4.—(X-ray) skiagram of lower dorsal and lumbar region of patient illustrating Type C of fibrositis ossificans ("osteoarthritis").

the disease has to be fairly advanced before any definite X-ray evidence of its presence can be detected. A comparison of these skiagrams and photographs shows this. How much more difficult, therefore, it must be to see the earliest signs in the living subject.

The *second* point, which is so interesting, is that even a cursory examination of the lateral borders of the diseased spines shows that all the pathological structure is independent of the bony constituent of the segments—the outlines of the normal bony vertebræ being more opaque show clearly through the other

shadows. There is, therefore, no true bony proliferation of the vertebræ, such as occurs in the other parts of the body, but the whole condition arises from a deposition of calcium salts in the lateral borders of the anterior common ligament, which gradually may spread and fuse over the whole of the anterior surface. It is, therefore, not really an osteo-arthritis, but a fibrositis ossificans. To explain the difference I produce an X-ray picture taken of an osteo-arthritic hip-joint split longitudinally. In this case true bone is present, this being, therefore, in the nature of an ossifying ecchondrosis.

If, as seems at present, the commonly accepted toxic theory as regards the causation of this disease be true, how is it possible to explain the differences between the two types A and B, the proliferating and the erosive, respectively. My suggestion put forward tentatively is that the explanation mainly rests upon the well-known fact that in all osteo-arthritic conditions there occurs shortly after the outset of the disease a considerable atrophy of the muscles surrounding the parts. The ligaments also become slackened, with the result that in all situations where gravity plays its part the bony segments of the joint tend to approximate, and, therefore, pressure is put upon the intervening cartilage. This is well illustrated in the case of the knee-joint, and one of the most successful methods of treatment consists in toning up the muscles around the joint by means of graduated contractions, with the result that when the muscles recover the pressure is taken off the intervening fibro-cartilage, and the pain and disability are lessened or disappear, although, of course, the pathological structures in the joint are unaffected. Before this pressure effect is produced, ossifying changes have already begun in the anterior common ligament, and it simply depends upon whether the ossifying process has gone far enough to produce a buttress-like effect between the two affected vertebræ before cartilaginous erosion appears or not. If, as commonly happens, this, so to speak, protective process has advanced far enough the disc is protected from pressure effects, and then we have Type A. If the anterior common ligament is unaffected, then we have a constant pressure on the disc or discs resulting in their gradual erosion and an approximating effect upon the vertebræ affected.

Sometimes both processes occur, and Type C is one in which erosion has taken place, and the buttress effect has come on too late to save the partial destruction of the disc.

In the case of other joints—the knee-joint for instance—in addition to the ecchondrosis, it seems probable that a slight ossification takes place in the ligament surrounding the joint, and thus produces the well marked curved spikes seen in advanced forms of this disease. But this does not proceed far enough to protect the cartilaginous erosion associated with this condition.

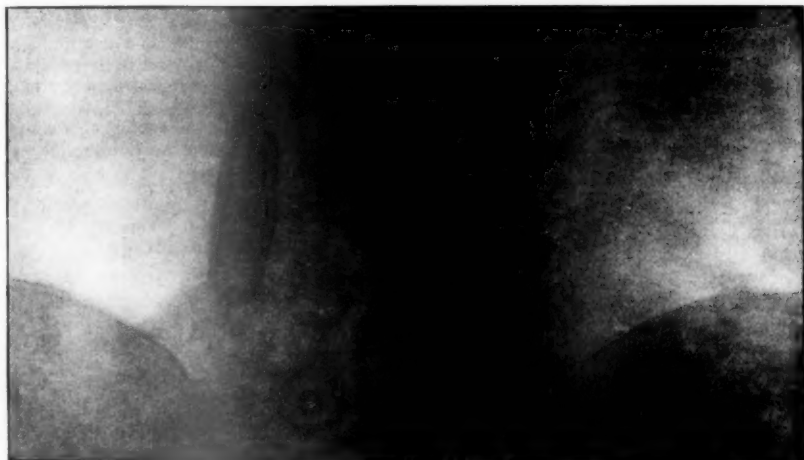
I am only showing four lantern slides, because it is difficult to see the different densities of the shadows involved on the screen, and I shall merely point out where the differentiation between true bone and the overlying fibrositis ossificans exists. I have examined a good many more spines than those exhibited here, but they all show the same condition.

I mentioned at the beginning that a new piece of apparatus has recently been put at our disposal by means of which we are enabled to obtain considerably better detail of the spine than has hitherto been possible, namely, the "Potter-Bucky Diaphragm," so-called after the two American radiologists who invented it, and the principle is simply this: X-rays are not only given off by the tube itself, but secondary radiations occur in the bones and tissues of the patient himself, which also affect the plate, and being scattered in all



directions have a fogging effect upon the detail. The longer the exposure, the more secondary radiations are produced, and this explains why obese patients are so difficult to X-ray satisfactorily—not only have they more thickness of tissue to produce these radiations, but also the time of exposure has to be considerably lengthened.

In this instrument a moving grid of thin lead strips is embedded vertically in wood and made to move at an even rate between the patient and the sensitized film during the time of the exposure. This serves to eliminate a greater portion of these deleterious rays, and now that we are able, by using two intensifying screens and fast films, to reduce considerably the time of the exposure, the increased exposure which would otherwise have been necessitated by the presence of the moving grid has been eliminated.<sup>1</sup>



This skiagram of the lumbar region of the back in Dr. Parkes Weber's case shows two "cigar-shaped" bodies, one on each side of the spinal column in the erector spinae muscle but not symmetrically situated.

Dr. F. PARKES WEBER thought that in diseases of the joints and spinal column when a true inflammatory causation was uncertain it was better to employ the termination "—osis" in preference to the termination "—itis," in fact, to speak of *arthrosis* and *spondylosis* instead of *arthritis* and *spondylitis*—just as some authorities distinguished conditions of "nephrosis" from conditions of "nephritis." Thus, there was Pierre Marie's type of spondylosis, in which not only did the vertebral column become rigid, but there was likewise a tendency to bony ankylosis of the shoulder and hip joints, and even the maxillary joint might be affected, as it was in a case which he (Dr. Weber) had described.<sup>2</sup> In a non-progressive case of spinal rigidity without involvement of the shoulder or hip-joints (cf. the Bechterew type), which he had seen, he believed that there had been a gonorrhœal element in the causation. Another case was most

<sup>1</sup> See also "Experiences with the Potter-Bucky Diaphragm," by Geoffrey Fildes, M.B., *Proc. Roy. Soc. Med.*, 1922, xv (Sect. Electro-Therap.), p. 7.

<sup>2</sup> F. Parkes Weber, *Proc. Roy. Soc. Med.*, 1914, vii (Clin. Sect.), p. 146.

puzzling. The patient, a man aged 37, had old chronic nephritis and a certain amount of kyphosis and stiffness in the vertebral column. In skiagrams of the lumbar and sacral regions besides some evidence of old vertebral disease two peculiar bodies could be seen, asymmetrically situated, one on each side of the vertebral column. By lateral Röntgen-ray examination, and by palpation, they appeared to be calcified or ossified masses and situated in the erector spinæ muscle, not directly adherent to the vertebral column. According to the history given, the patient had been under treatment at Paris in 1911 for a chancre on the penis, probably syphilitic, and had been given injections into the "lower part of the back." At first Dr. Weber had been inclined to explain these peculiar bodies as dried-up psoas abscesses, calcified and obsolete. But they were found to be in the erector spinæ and not in the psoas muscle, and now he believed them to represent the results of aseptic necrosis produced by intramuscular injections of salvarsan. In the early days of salvarsan therapy necroses from intramuscular injections undoubtedly sometimes occurred. Aseptic necrosis might occur without the extrusion of a slough, and in this case it seemed probable that the "cigar-shaped" bodies in question were local muscular necroses which had become encapsuled and partially calcified or ossified. On comparing a skiagram taken in 1917 with one taken in 1923 it was evident that the condition had remained stationary. By intramuscular injections of quinine a muscular necrosis might likewise be produced and might become encapsuled in a fibrous sheath, calcium salts being afterwards precipitated. This was illustrated by Mr. Denham Pinnock at the Section for Tropical Diseases, on June 12, 1922, when he demonstrated a cigar-shaped "tumour" removed from the gluteal region of a man, who had been treated in Salonika for malaria by intramuscular injections of quinine.

## Glandular Fever and Infective Mononucleosis.

By H. LETHEBY TIDY, M.D.

(ABSTRACT.)

[This paper will be published in full in the *Lancet*.]

GLANDULAR fever is an acute, infectious disease, principally of childhood, and is characterized by marked and rapid enlargement of the cervical glands and by a less constant enlargement of the spleen and other glands. The cervical glands may be slightly tender but are not acutely painful. The changes in the fauces are slight compared with the marked glandular enlargement. The temperature rises with the glandular enlargement to 103° F., or even 105° F., for a few days. The only definite complication is hæmorrhagic nephritis. Suppuration is very rare. Convalescence is usually slow, but recovery is finally complete and the mortality is negligible. The condition has no relation to mumps, whooping-cough or other diseases.

During the last two years several articles have been published on a condition of transient lymphocytosis under the title of "Infective Mononucleosis." The lymphocytosis is both relative and absolute. The percentage may rise to 95.

The clinical manifestations of these cases are indistinguishable from those of glandular fever, and the question of their identity arises.

In May, 1921, an epidemic of glandular fever in a boys' school came under observation. Out of a total of thirty boys, twenty-four had definite attacks. The epidemic was mild but many of the cases were typical. The blood was examined in eighteen cases. There was some difficulty about the examination as it was necessary to avoid any appearance of experimenting on the boys.

[April 24, 1923.]

Also, it was not realized that the lymphocytosis may take time to develop and may be very transient. The blood counts, however, suggest that an absolute lymphocytosis is a normal occurrence in glandular fever. It is not invariable, for in one case in which suppuration was feared a polynuclear leucocytosis was present.

Sporadic cases which have been under observation have shown similar blood changes and the same clinical characteristics.

The following questions arise:—

(1) *Is Glandular Fever a Clinical Entity?*—The description given of glandular fever applies equally to Pfeiffer's original cases, to the numerous cases recorded in the literature, to the cases in the epidemic, and to the sporadic cases which have been seen. It is also accurate for cases recorded as "infective mononucleosis," for many cases recorded as "acute lymphoid leukaemia with recovery," and under similar titles. The slowness of the changes in the fauces compared with the marked glandular enlargement is especially striking. The symptom-complex is characteristic, distinct from other diseases and sufficient to establish glandular fever as a clinical entity.

(2) *Are Glandular Fever and Infective Mononucleosis identical?*—As stated above no distinction can be drawn between the two conditions, either on clinical grounds or on the examination of the blood. It is consequently considered that glandular fever and infective mononucleosis are identical, and that an absolute lymphocytosis is a normal, though not invariable, occurrence in glandular fever.

(3) *Nature of the Mononuclear Cells.*—Usually the cells are not quite characteristic either of small or large lymphocytes. The nucleus is less regular, is frequently notched, and often eccentric. The amount of protoplasm is greater than is normal in small lymphocytes and stains more deeply. These abnormalities have been carefully described by writers on infective mononucleosis. The cells would appear to be immature lymphocytes.

(4) *Relation to Other Diseases.*—(a) In many of the recorded cases the patients have previously or subsequently had whooping-cough. The same statement applies to mumps, scarlet fever, and measles. (b) *Acute leukaemia:* There is no evidence that this is infectious. (c) *Tonsillitis and septic infections:* The changes in the fauces are characteristically slight in glandular fever, and often do not exceed a slight general redness and dryness. Definite tonsillitis is unusual. There is no evidence that oral sepsis or dental caries has any connexion with glandular fever.

Sepsis is frequently stated to be a rare but occasional cause of absolute lymphocytosis, but a study of the literature suggests that the recorded cases are either acute leukaemia or glandular fever, and there appears to be no evidence that sepsis does produce lymphocytosis.

#### DIAGNOSIS.

The diagnosis is usually simple if glandular fever is borne in mind. It is occasionally confused with acute leukaemia if the blood is examined, but the rapid recovery soon casts doubt on this diagnosis. The usual absence of severe constitutional symptoms and the general clinical condition makes a differential diagnosis far from difficult. In the absence of a blood count the condition is most often mistaken for mumps or "aberrant mumps," a mistake which in effect is of little importance. The diagnosis of acute tuberculous adenitis is occasionally made, and such an error may have serious consequences.

CONCLUSIONS.

- (1) Glandular fever is a clinical entity.
- (2) An absolute lymphocytosis is a normal occurrence.
- (3) Glandular fever and infective mononucleosis are identical.
- (4) Recovery is permanent, and there is no relation to leukæmia, Hodgkin's disease, tuberculosis, or other conditions.
- (5) There is no evidence that sepsis is a cause of absolute lymphocytosis.

## Section of Medicine.

President—Dr. G. NEWTON PITT, O.B.E.

### Case of Acquired Chronic Hæmolytic (Acholuric) Jaundice, seen Fifteen Years ago, with a Blood Picture at that time resembling one of Pernicious Anæmia.

By F. PARKES WEBER, M.D.

THE patient is a woman, now aged 53, who was first admitted to hospital under my care in January, 1908, when she was aged 37.<sup>1</sup> According to the history given at that time she had been healthy till three years previously, when she began to look pale. About a year later she had an attack of jaundice, with pain in the right side, and afterwards gradually grew weaker and paler, and short of breath on exertion. Menstruation became scanty, and for a time there was complete amenorrhœa. She suffered from recurrent epistaxis and was troubled with coldness and numbness of the hands and feet; sometimes there was a sensation of "pins and needles" in the fingers, which would "go blue."<sup>2</sup>

After admission to hospital in January, 1908, the condition noted was the following: The skin and conjunctivæ were yellowish. There was no pruritus or xanthoma. The legs were œdematous. The heart was slightly dilated, and there was a faint systolic murmur, apparently not due to valvular disease. The liver and spleen were both evenly enlarged. The liver extended two or three finger-breadths below the ribs, and the spleen, which was hard, reached almost to the anterior superior iliac spine. The condition of the patient's mouth was not bad, though she had lost many of her teeth. Occasionally there was slight bleeding from the gums. Attacks of epistaxis were frequent. Ophthalmoscopic examination (Dr. C. Markus) showed numerous bright red, round, retinal hæmorrhages in both eyes, chiefly in the upper halves of the fundi. The urine, of specific gravity 1013, contained albumin (1 per mille by Esbach's tube on admission), but no tube-casts. A later note of the urine stated it to be of specific gravity about 1015, of deep orange colour, free from albumin and sugar, and giving no Gmelin's reaction for bile pigment, but containing excess of urobilin. The fæces were never acholic. Examination

<sup>1</sup> I showed the case at the Medical Society of London on April 13, 1908, February 8, 1909, and January 27, 1913 (*Trans. Med. Soc. Lond.*, 1913, xxxvi, p. 360). I also described the case in full in the *Amer. Journ. Med. Sci.*, Philad., 1909, cxxxviii, p. 24, and published a sequel in the *Practitioner*, Lond., 1913, xc, p. 811.

<sup>2</sup> I would here note that even more decided nervous troubles and actual combined degeneration of the spinal cord may occur not only in pernicious anæmia but also in various anæmic and cachectic conditions. Salomon (*Gesellsch. f. inn. Med. u. Kinderheilk. zu Wien*, January 29, 1914) showed a man, aged 32, with hæmolytic jaundice and combined degeneration of the spinal cord.

of the gastric contents after a test-breakfast (May, 1908), showed complete absence of free hydrochloric acid.

Examination of her blood on January 31, 1908, gave the following result: Hæmoglobin (by Haldane's method), 18 per cent. of the normal; red cells, only 900,000 in the cubic millimetre of blood; white corpuscles, 6,000; colour index, 1. The differential count of the white cells gave: Lymphocytes, 45.6 per cent.; transitionals, 4.8 per cent.; large mononuclears, 2.4; neutrophil polymorphonuclears, 46; eosinophil, 0.8; mast cell, 0.4. During the count of 500 white cells Dr. A. E. Boycott saw no ordinary normoblasts, but eight typical megaloblasts (giganto-blasts) of the pernicious anæmia type, and sixteen smaller nucleated red cells, resembling the typical megaloblasts except in regard to their relatively small size. There were a few polychromatophilic red cells and many punctate basophilic red cells. Decided anisocytosis and poikilocytosis were present, as in pernicious anæmia. The blood-picture at that time was one of typical "megaloblastic degeneration"—I would prefer to say "megaloblastic regeneration" or "megaloblastic regenerative reaction"<sup>1</sup>—but a striking feature was the association of chronic acholuric jaundice, and the case likewise differed from pernicious anæmia in regard to the enlargement of the spleen and liver. I regarded the case at first as a variety of pernicious anæmia with unusual enlargement of the spleen and liver, but afterwards I came to the conclusion that it was one of acquired chronic hæmolytic (acholuric) jaundice, with gastric achlorhydria and a blood-picture suggestive of pernicious anæmia. The resistance of the patient's erythrocytes to hæmolysis, as tested with graduated hypotonic sodium chloride solutions, was less than that in various healthy persons used as controls.

In the treatment of the case the arsenical preparation known as atoxyl was especially employed; but the great improvement which ultimately followed, could not with certainty be attributed to the atoxyl, as the period of the most decided improvement commenced a considerable time after the atoxyl treatment had been discontinued.

In December, 1908, the blood showed nothing abnormal beyond slight anæmia. She became free from jaundice, fever and œdema. The liver could no longer be felt enlarged. The retinal hæmorrhages cleared up. The condition remained fairly good, and the patient was able to do ordinary work as a domestic servant. On July 17, 1911, the red cells numbered 5,330,000 to the cubic millimetre of blood.

Several features of the patient's illness up to the summer of 1909 require special mention and consideration.

*Abdominal Pain.*—At various times the patient complained of pain or tenderness in the upper part of the abdomen, sometimes in the region of the liver, sometimes associated with fever. An attack about the middle of August, 1908, was associated with vomiting and temporary increase of the icteric tinge; a short attack in December, 1908, was accompanied by a sudden rise of temperature to 103° F. Attacks of this kind are well known in hæmolytic jaundice and represent temporary exacerbations of the disease. They may be mistaken for attacks of cholelithiasis, and in fact gall-stones have been found in some cases.

*Jaundice.*—While under observation the patient was never deeply jaundiced, but the conjunctivæ for a long while were distinctly yellow. With the improve-

<sup>1</sup> That is to say, the blood-picture of so-called "megaloblastic degeneration" must be interpreted as representing an automatic attempt at regeneration.

ment in the general condition in November, 1908, the jaundice disappeared. With a single doubtful exception (during a temporary exacerbation of the jaundice), bile pigment was never present in the urine; the fæces were never acholic.

*Temperature.*—At first there were recurrent periods of moderate fever, but after September, 1908, there was hardly any fever. On December 13, 1908, a temperature of 103° F. was noted in association with abdominal pain (see above, under *Abdominal pain*).

About June, 1912, the patient commenced to suffer from interstitial keratitis of the right eye, which was evidently regarded at an eye hospital as of syphilitic origin. When seen at the end of 1912 and in the early part of 1913 she was slightly anæmic, but microscopical examination of blood films showed nothing abnormal. There was no excessive fragility of the erythrocytes towards hypotonic saline solutions. The liver was apparently not enlarged. The spleen could just be felt below the costal margin. The conjunctivæ had a slight yellowish tinge, and the urine (December, 1912) showed some excess of urobilin. Her blood-serum on one occasion (January, 1913), gave a positive, but on two other occasions about that time a negative, Wassermann reaction.

On May 8, 1923, the patient was readmitted under my care with the history that since I last saw her in 1913 she had remained in good health till the end of 1922, when, possibly as a result of excessive work (as a monthly nurse, cook, &c.), she began to suffer from a feeling of weakness, a tendency to cardiac palpitation, and giddiness on exertion; she had also noticed pallor and yellowness of her face. The menopause had occurred about 1921, when she was aged 52. On readmission I find her looking obviously anæmic, with light icteric coloration of her skin and sclerotics. The spleen is moderately enlarged, and the liver (not very hard to palpation) reaches down to the umbilical level. The temperature is occasionally very slightly raised; pulse, 66-80; respiration, 16-20; brachial systolic blood-pressure, 115-140 mm. Hg. The fundi of the eyes appear normal, but there are posterior synechiæ in the right eye from earlier plastic iritis (Dr. C. Markus). The gastric contents after a test-breakfast show complete absence of free hydrochloric acid and of pepsin (*achylia gastrica*). The blood serum (May 10, 1923) gives a completely negative Wassermann reaction. The fæces are, if anything, rather deeper coloured than average fæces. The urine is of a reddish-orange colour, free from albumin, sugar and bilirubin, but showing excess of urobilin and urobilinogen. It is free from bile acids, according to surface tension estimations by Dr. J. W. McNee and Mr. E. A. B. Pritchard.

For the present blood examinations I am indebted to Dr. G. Welseh. The blood count (May 9, 1923) gives: Erythrocytes, 1,800,000 per cubic millimetre of blood; hæmoglobin, 40 per cent.; colour index, 1.1; white cells, 6,100 per cubic millimetre of blood. The differential count of 300 white cells gives: Neutrophil polymorphs, 74 per cent.; lymphocytes, 24.5 per cent.; no transitionals or large mononuclears; mast cells, 1.5 per cent.; no eosinophils. In regard to the red cells there is much anisocytosis; the macrocytes are especially numerous, and show polychromatophilia and decided poikilocytosis. Whilst making the differential count of white cells three nucleated red cells were observed: two of these were normoblasts with polychromatophilic cytoplasm, and the other was a large megaloblast with a large ("loose") nucleus and polychromatophilic cytoplasm. The resistance of the erythrocytes towards hæmolysis was estimated (May 10, 1923), by adding drops of the whole blood



to graduated hypotonic sodium chloride solutions. The hæmolysis was found to commence with a 0.52 per cent. solution, and to be complete with a 0.42 per cent. solution. The resistance of the erythrocytes is therefore definitely below the normal standard, that is to say, their fragility is somewhat excessive, as it is in cases of congenital and acquired hæmolytic jaundice.

Dr. J. W. McNee (June 1, 1923) kindly examined the blood for bilirubin and bile acids. The blood serum was yellowish and opalescent (physiological lipæmia, owing to a recently taken meal). It gave a negative *direct*, but a positive *indirect* Hijmans van den Bergh reaction for bilirubin, equivalent to  $5\frac{1}{2}$  van den Bergh units (or 1 in 36,364). It was free from bile acids, according to surface tension estimations (Mr. E. A. B. Pritchard).

The case is one of hæmolytic (acholuric) jaundice, apparently acquired. The old plastic iritis and interstitial keratitis in the right eye suggest that the patient may likewise have had syphilis. I have recently examined her only living child, a sailor, aged 25, who appears to be healthy, and presents no signs of hæmolytic jaundice or syphilis. After the birth of that son she never became pregnant again, but before his birth she now tells me that she had: (1) a female child, who died at 1 year 9 months of "convulsions," (2) a miscarriage, and (3) a stillborn child.

At first, in January, 1908, I temporarily mistook the case for one of pernicious anæmia, and it is interesting to note that the blood picture now again (May, 1923) is of the pernicious anæmia type. In cases of congenital hæmolytic jaundice poikilocytosis is usually not a feature of the blood-picture, but there is generally great anisocytosis, with predominance of *apparent microcytes*. These "microcytes" are not true microcytes, as they are not really small; they have been shown by Naegeli and others to be only apparently small, because they have a relatively small diameter and a relatively small circumference; but, in regard to their total volume, they are really large, owing to their globular or ball-like shape ("Kugelzellen"). In some cases of hæmolytic jaundice (especially acquired cases), like the present one, there seems to be a tendency for the blood-picture, during exacerbations of the disease, to assume the "pernicious anæmia type," with poikilocytosis and megaloblasts, and with predominance of "macrocytes," instead of the ball-cells ("Kugelzellen") of Naegeli.

It is possible that the patient's present exacerbation may be due to excessive work and fatigue. In regard to the injurious influence of mental strain and physical fatigue in cases of hæmolytic jaundice I may mention that one of the family (Ernest T.) of congenital hæmolytic jaundice reported by Dr. Dorner and myself in 1910,<sup>1</sup> who was at that time aged 12, and only slightly affected, had later on to undergo great strain as an artilleryman during the Great War. Under the strain, after a considerable time, he broke down with an exacerbation of the disease. He became extremely anæmic, and his splenomegaly greatly increased, as Dr. J. H. Drysdale kindly showed me in St. Bartholomew's Hospital, where splenectomy was successfully performed in 1922. That patient's paternal grandfather, though jaundiced all his life, lived to 76 years (not 70 years, as, owing to an error in copying, we stated in the account published in 1910), and both this and other published data prove that patients with hæmolytic icterus may sometimes under ordinary circumstances live to a good age, like normal persons, even though splenectomy is never performed.

<sup>1</sup> F. Parkes Weber and G. Dorner, "Four Cases of Congenital Acholuric (Hæmolytic) Jaundice in One Family," *Lancet*, Lond., 1910, i, pp. 227-232.

The good results obtained by splenectomy (when successfully performed) in cases of hæmolytic icterus seem to be due not only to the removal of the acknowledged hæmolytic action of the spleen, but likewise to the removal of some subtle influence of the spleen on the bone-marrow in these cases, owing to which influence the bone-marrow produces abnormal erythrocytes with low resistance towards hæmolysis (cf. recent literature on the subject).<sup>1</sup>

*Note.*—Since the last above-recorded blood count was made the patient has had a course of iron and arsenic treatment.

#### DISCUSSION.

Dr. WILLIAM HUNTER said that the case now recorded by Dr. Parkes Weber lent itself to discussion in its many relations—the characters of the hæmolytic disease termed pernicious anæmia, those of the pathological condition termed acholuric jaundice, and the blood changes associated with these two conditions. But the most important point appeared to be the nature of the case itself; and it was to this point that he would confine his remarks, basing them on his long experience and special interest in the subjects of anæmia and jaundice respectively.

*The Case.*—He had seen the patient, a woman aged 53, obviously anemic, presenting a slight lemon tint of sclerotics and skin, the appearance so characteristic of the hæmolytic disease pernicious anæmia. The other features in the case relating to degree and character of blood-changes, character of the pigment changes in the urine, and the presence of achylia gastrica, were also those exhibited in pernicious anæmia. In addition to these usual criteria of the disease, he had applied the tests of his newer diagnostic criteria, viz., (1) the existence of *glossitic features*, viz., the history or presence of symptoms or lesions of sore tongue; and (2) the characters of *seasonal incidence* connected with the original onset or the subsequent relapses of the disease, viz., autumnal in 90 per cent. of onsets and relapses. These he had recently described before this Section.<sup>2</sup> This latter feature of seasonal incidence he had found so marked and constant, that the year of disease for pernicious anæmia dated in his observations not from January to December, but from July to June. In applying these criteria to the present case, and to her present illness, quite apart from her earlier illness twelve years ago, the patient stated that it begun in the autumn of last year, and that it was manifested by anæmia and yellowness. This description of onset of this disease and of its relapses was one that in his experience applied to every case of the disease. As the report of the case showed, it was exactly the features of the onset of the disease in 1905-06 before the case came under observation in January, 1908. As regards the glossitic features of the case, these were the only features not mentioned or noted in the report. On being asked whether she had any trouble with her tongue, the patient at once stated that at the onset of her present attack last autumn her tongue had been so sore that she could hardly drink even warm water. And yet the tongue at the present time looked quite normal—a feature which gave place at times to definite evidences of lesions, as he had fully described.

*Nature of the Case.*—On the characters of the case as now presented, he had therefore no doubt in his mind that it was definitely pernicious anæmia, presenting the usual hæmolytic features of that disease as originally described by him (Dr. Hunter) in 1890, and perpetually emphasized by him since. He therefore concluded that if the case was now one entitled to the name of, and to be regarded as one of, acholuric jaundice and not as pernicious anæmia, then every case of that disease he had seen, and also every case of that disease seen by others, was not pernicious anæmia, but acholuric jaundice. For, as he had described in his first studies of the urinary pigment changes in this disease in 1888, the most striking feature of the great hæmolysis marking the disease

<sup>1</sup> The excessive hæmolysis, due to defective erythrocytes, possibly leads to increased production by the bone-marrow of the defective type of erythrocytes in question—a "vicious circle," which to some extent can be "broken" by splenectomy.—F. P. W.

<sup>2</sup> *Proceedings*, 1922-23, xvi (Sect. Med.), pp. 1-42.

was the absence of bile pigment from the urine ("acholuria"), however great the degree of yellowness and apparent jaundice might be.

*Nature of the First Illness.*—As had been made clear by the full details given by Dr. Parkes Weber its features were practically identical with those of the patient's illness at the present time. Here again he desired to state that if he could form a definite opinion of its character at that time, it was due largely to the fact that Dr. Parkes Weber reported the facts of the case so fully. He had no doubt as to its nature, judging from the history of mode of onset, clinical and blood features presented, and subsequent progress of the case. There was good recovery early in 1908, followed by a slight threatening of relapse between August and December of that year (seasonal exacerbation); followed again by a higher degree of recovery during 1909-10, and greater part of 1911, till the late autumn when there was another definite threatening of relapse shown by anæmia and yellowness and urobilinuria; then a still more prolonged recovery lasting for a period of ten years; till this in turn was followed by the decided relapse late in the autumn of 1922 from which the patient was now suffering. This case from its onset was thus one of the hæmolytic disease pernicious anæmia, running the prolonged course which, as he had recently described,<sup>1</sup> a certain number of cases of that disease were capable of doing.

He had the records of a precisely similar case starting in 1908, seen in 1911, with red cells 34 per cent. and definite history of sore tongue; good recovery till late in 1912, when he had a severe relapse marked by loss of 2 st. in weight; once more a still better recovery lasting for six years till 1920 when again he had a severe relapse with blood count down to 30 per cent. of red cells, this period being twelve years after the original onset of his disease. The patient was afterwards continuously ill for two years till he died in August, 1922. The case was seen in consultation with Dr. Henry Carmichael, of Wandsworth. The original diagnosis of the case as one of pernicious anæmia made by him (Dr. Hunter) in 1911 was independently confirmed by Dr. (now Sir Frederick) Mott in the same year. The duration of the case from first to last was thus fourteen years.

The case thus very closely resembled the one now under consideration as regards its duration and its variations. But it resembled it even more closely in the other feature which Dr. Parkes Weber stated induced him to change his original diagnosis of pernicious anæmia and to regard his case as one of acholuric jaundice, namely, the persistence of a marked degree of enlargement of the liver and spleen, when the case was first seen in 1908—an enlargement which subsequently disappeared, and had again recurred in the patient's present attack. The case he (Dr. Hunter) had just reported presented a similar enlargement of the liver and spleen in his last illness (1920-22), the enlargement of the spleen being so marked that one observer was inclined to regard the case as one of Banti's disease which it was not.

In his experience, more or less enlargement of the liver and spleen was the rule whenever the hæmolytic disease was active, this enlargement passing off when the disease became quiescent. In rare cases it might be very great, the spleen extending down to the iliac crest as in the above case; and he had a precisely similar case under his care at the present time. His post-mortem experience confirmed the existence of this great enlargement, weights of 15 to 24 oz. being not uncommon in cases of the disease, instead of the normal 5 to 6 oz., and the liver weighing 6 lb. 5 oz., instead of the normal 3 to 4 lb.

*Conclusion.*—He thus concluded that the features and course of the case in its earlier stages, as at the present time, were those of pernicious anæmia.

*Post-mortem Character of a Case of Pernicious Anæmia dying in State of Jaundice.*—The final decision as to the character of the anæmia presenting marked features of jaundice could be arrived at if a patient died in this stage. He had had such a case in 1908—a man aged 48, admitted February 1 with what appeared acute and severe ordinary jaundice, but recognized by him on his first seeing the case to be something different, and to be suggestive of pernicious anæmia: and this it proved to be. The patient had a two months' history of illness marked by weakness and

<sup>1</sup> *Proceedings*, 1922, xvi (Sect. Med.), pp. 1-42.

vomiting and this was followed by a three weeks' history of jaundice. He was intensely anæmic beneath his jaundiced appearance, which was intense, his blood showing 21 per cent. of red cells, with colour index 1.32; leucocytes 3,400, with megaloblasts 2 per cent. of the number of leucocytes. By February 13 the jaundice had passed off, and was replaced by lemon tint. But the temperature began to rise, at first to 100.6° F., then to 104°; the patient became comatose and died on February 16.

The post-mortem showed: Liver not enlarged; spleen large and soft and of a deep chocolate colour; weight 21 oz. The liver gave a marked reaction of iron, and chemical analysis of the liver, kidney and spleen showed a large excess of iron, viz., 0.278 per cent. of dry residue in the liver as compared with 0.080 in health; 0.107 per cent. in the kidney as compared with 0.004 in health; and 0.140 per cent. in the spleen, as compared with 0.090 in health.

The character of the case marked by this intense jaundice was thus made clear as being a most intense manifestation of the hæmolytic disease called pernicious anæmia. And such in his final judgment was the character of the case now described and entitled as one of acholuric jaundice. To have the great individuality of that remarkable hæmolytic disease—as in its total assemblage of features and lesions it presented itself to him (Dr. Hunter)—altogether lost and hidden under the title of acholuric jaundice, would in his opinion put back the clock of this interpretation of this disease, already a matter of sufficient difficulty. Every feature of the disease would in turn be written of afresh and ascribed to acholuric jaundice or to some form or other of hæmolytic jaundice. This result was already foreshadowed by the case, mentioned in Dr. Parkes Weber's paper, of the German observer who had reported that of a man, aged 32, with hæmolytic jaundice and combined degeneration of the cord—the lesion of the cord most closely and intimately associated with pernicious anæmia.

It was in the hope and with the strong desire of preventing the inevitable confusion that would thereby be caused, that he had given this detailed consideration to the case so fully and clearly recorded by Dr. Parkes Weber. He had only been able to do this through the courtesy of Dr. Parkes Weber who had kindly allowed him to see the manuscript of his paper and thereby enabled him to obtain accurate and full details of the case before expressing a final opinion about it.

*Long Duration of the Case.*—Even if the case were now one of the hæmolytic disease "pernicious" anæmia—the disease more stringently connoted by the title of "Addisonian anæmia," still more by the title of "glossitic hæmolytic anæmia," given to it by him (Dr. Hunter)—the long duration over a period of eighteen years from its onset in 1905, and the first severe attack at the end of 1907, up to the present time—1923—seemed to be quite inconsistent with the character of an anæmia entitled to be called "pernicious," a name implying a prognostic duration of one, two or, at longest, three years. This was true as regards the title "pernicious"; but the actual facts of the disease concerned were stronger than the unhappy and ill-omened name it bore—a matter that he (Dr. Hunter) had recently most strongly emphasized (*British Medical Journal*, March, 1922). For in individual cases patients could, in his experience, recover, and resist their disease for periods of six, seven, ten, thirteen, and even up to twenty years, as in the case he had recently recorded before this Section. They could at the end of such lengthy periods demonstrate the original character by having relapses presenting the identical features of the original attack, as in the case of his own he had just described. The case now recorded by Dr. Parkes Weber was in his (Dr. Hunter's) opinion of a similar character.

Surgeon Rear-Admiral BETT ventured to mention a case which came under his observation at a naval hospital in 1919. Patient had been in various hospitals in 1907, 1913, 1915, 1916, 1917, with attacks of jaundice with enlarged spleen and anæmia and normal urine. Red blood counts had varied from 1,280,000 to 5,437,500. No family history of jaundice or anæmia. On admission: Urine; spectroscopic examination showed absence of urobilin; conjunctivæ tinged with yellow, skin very slightly. Red cells, 3,250,000; no leucocytosis. Spleen felt two fingers below costal margin. Liver not enlarged. Slight puffiness of hands and upper lip. Patient said he had observed this several times before. No epistaxis; no tendency to hæmorrhage of any kind;

suffers from headache, nausea and sometimes vomiting. (In 1915 the attack followed a severe contusion.) Patient improved very much with rest and on discharge the count showed 4,950,000 red cells. Operation was discussed; not recommended—and declined. This patient had another similar attack afterwards after working very hard in his garden; again made good recovery and when seen recently was in good health. Conjunctivæ never entirely free from jaundice.

Dr. F. J. POYNTON said that he approached the subjects introduced by Dr. Parkes Weber from the point of view of congenital family cholemia. In the early days of the recognition of this disease he had shown groups of cases and had pointed out that a case might at one time become entirely jaundiced, at another profoundly anæmic. When jaundice was in the ascendant the liver enlarged. When anemia was to the fore the spleen enlarged, and the red cell count might fall below 1,000,000 per cubic millimetre. Turning to pernicious anæmia they recognized cases in which the anemia was very profound and others were seen in which there was considerable acholuric jaundice, varying in intensity and associated with blood crises. Thus in the two diseases the parallel events were seen, but a distinction had been drawn between them upon which he would much like to hear the opinions of the meeting. It was this; in congenital family cholemia and in acquired family cholemia the red cells showed an undue fragility to saline solutions, but not so the red cells in pernicious anæmia. What value was to be attached to this distinction? If it was of fundamental importance then Dr. Weber's case could not be called one of pernicious anæmia. It appeared to him that this question of the fragility was one of the greatest interest and scientific importance if it could be relied upon in differential diagnosis.

Dr. THURSFIELD said that the standard of fragility of the erythrocytes to dilutions of saline was so constant and unvarying under all physiological conditions that any variations from the normal should be accepted as an indication of disease. Increase of fragility was, however, so rare and uncommon that with present knowledge it could be affirmed to exist only in an extremely limited class of cases: and acholuric jaundice, whether congenital or acquired, easily took the first place. The picture of the blood on the other hand notoriously varied and there was more than one condition in which the picture which was associated in their minds with pernicious anæmia was reproduced in every detail without the presence of this disease. Moreover, in all the undoubted cases of Addisonian anemia which he (Dr. Thursfield) had examined for fragility the standard had been either normal or subnormal, that is the resistance to hæmolysis had been increased. Holding these views Dr. Parkes Weber's case, in which, though the blood-picture was that of pernicious anæmia, yet the fragility of the erythrocytes was definitely more pronounced than in a normal state, he (the speaker) was obliged to consider that the patient was suffering from an acquired acholuric jaundice, and not from Addisonian anemia.

Dr. H. L. TIDY said that he doubted whether there was really sufficient knowledge at the present time to decide in what class this case should be placed. They were learning now that a blood-picture resembling pernicious anæmia occurred in conditions differing from those to which this name had been generally applied. It had long been recognized that it might occur in infections with *Dibothriocephalus latus*. It had more recently been recognized as occurring in sprue, and lately it was found to be present in Hurst's case of Addison's disease which was treated by a suprarenal graft. It had also been seen occasionally in other circumstances. The following instance had been under his own observation:—

A boy, who was now 17 years of age, had suffered from early childhood with pain and swelling of the joints. At the age of 14 he became acutely ill, and was found to have a spleen below the umbilicus and a blood-picture resembling pernicious anæmia. The diagnosis of different physicians had varied between pernicious anæmia and Still's disease. He (Dr. Tidy) saw him about six months later when he had greatly improved, and the diagnosis of pernicious anæmia would not have been made on the blood count. He could not find any information as to the blood changes in Still's disease. Two points might be noted: First, the fragility of this patient's red cells was greater than normal; and secondly, there was a definite family history of splenomegaly. His grandmother, who was still living, had had an enlarged spleen for many years, and his great-grandmother had an enlarged spleen and jaundice; his mother had also had jaundice and gall stones. He would not regard this case as pernicious anæmia but as a primary bone marrow error.

With regard to the fragility of red cells, he (Dr. Tidy) agreed with Dr. Poynton and Dr. Thursfield that an increase, so far as was known at present, was confined to acholuric jaundice, and did not occur in pernicious anæmia. In ordinary forms of jaundice the resistance was greater than normal. In Dr. Parkes Weber's case they would note that the fragility was increased, and also that the spleen was distinctly enlarged, reaching to the umbilicus—a very unusual size in pernicious anæmia. For these reasons the case should be considered to be one of acholuric jaundice and not of pernicious anæmia.

Dr. PARKES WEBER (in reply) maintained that his case corresponded in all essential points to one of hæmolytic (acholuric) jaundice. The great difficulty in such a case was to be sure that it was not really one of previously latent *congenital hæmolytic jaundice*. He had no doubt that formerly cases of hæmolytic (acholuric) jaundice, during exacerbations with the blood-picture of what he would call "megaloblastic regenerative reaction," had been occasionally diagnosed as cases of pernicious anæmia, especially when little or no obvious icteric coloration of the face and sclerotics was present. In regard to Dr. Tidy's cases he suggested that the boy was an example of "infective rheumatoid arthritis of Still's type," occurring in a subject of latent congenital hæmolytic icterus and giving rise to an anæmic exacerbation of the latter disease. Probably another case referred to by Dr. Tidy was a true one of *partial aplastic anæmia*, in which only part of the bone marrow underwent *aplastic degeneration*, the other portion of the bone marrow undergoing a *regenerative reaction* of the "pernicious anæmia type." The good effects of splenectomy, when successfully performed, in cases of hæmolytic (acholuric) jaundice could only be explained by the removal of the hæmolytic function of the spleen, *together with* the removal of some influence of the spleen on the bone marrow which had caused the latter to produce a greater or lesser percentage of erythrocytes abnormally fragile towards hypotonic saline solutions.

### A Rare Case of Congenital Non-familial Jaundice, without Enlargement of Liver or Spleen, in an otherwise Healthy Man, aged 56.

By F. PARKES WEBER, M.D.

I FIRST brought forward this case at the Clinical Section of the Royal Society of Medicine on February 9, 1917.<sup>1</sup> G. T. D., an Englishman, was at that time aged 50. He was an active, well-built man, of average general nutrition, and (according to his mother) he had been yellow on birth, and had remained more or less yellow ever since then. The yellowness affected the sclerotics as well as the skin. On the whole he seemed to have enjoyed unusually good health, and had not been subject to headaches, "bilious attacks," or cutaneous pruritus. He was sure that his fæces and urine had been of ordinary colour, like those of normal persons. He had had no xanthoma, not even xanthelasma palpebrarum. His friends told him that his yellow coloration was more marked at some times than at others. There was nothing special in his family history; none of his four children and none of his other relatives were known ever to have had a similar yellow appearance. I could find nothing abnormal by ordinary examination of the thoracic and abdominal viscera. There was no enlargement of the spleen or liver or superficial lymphatic glands. His urine was clear, of pale yellow colour and acid

<sup>1</sup> F. Parkes Weber, *Proc. Roy. Soc. Med.*, 1917, x (Clin. Sect.), p. 13.



reaction; the ordinary chemical reactions were negative for albumin, sugar and bilirubin, and for excess of urobilin, urobilinogen and indican. In regard to the examinations of the blood and blood-serum I was much indebted to Dr. H. Schmidt. The hæmoglobin was 105 per cent.; erythrocytes, 6,400,000 to the cubic millimetre of blood; white cells, 5,710 (of which about 80 per cent. were polymorphonuclear neutrophils). Microscopic examination of stained blood-films showed nothing special, no nucleated red cells, no poikilocytosis, no polychromatophilia and no marked anisocytosis. The blood-serum was more deeply coloured than that of most persons. The resistance of the erythrocytes to hæmolysis was tested by adding drops of the patient's defibrinated blood to graduated hypotonic solutions of sodium chloride. It was found to be within normal limits, hæmolysis commencing with a solution of 0.48 per cent. and being complete with a solution of 0.34 per cent. The blood serum gave (January, 1917) a negative Wassermann reaction for syphilis.

Since then the man, who is now aged 56, has kept in excellent health, and I have more than once satisfied myself that no enlargement of the spleen or liver can be detected. He has slight deafness, doubtless due to otosclerosis, and has slight nystagmus. He has again assured me that he has never tended to suffer from constipation or from any kind of "biliousness," and that the yellowness has not been observed in any other member of his family. The sclerotics of his eyes are of a bright canary-yellow colour and I think that the average degree of icteric coloration has become deeper than it was in 1917. But his urine is clear, of pale yellow colour, acid, and free from albumin, sugar and bilirubin, and from any excess of urobilin or urobilinogen; specific gravity (February 5, 1923), 1025. On May 22 the urine was carefully examined by Dr. J. W. McNee, of University College Hospital, and was proved to be free from bile acids.

At one time I thought that the yellow colour of the patient must be due to some pigment other than bilirubin,<sup>1</sup> but Dr. J. W. McNee has kindly examined the blood by the Hijmans van den Bergh method. He finds (April 30, 1923) that the patient's blood serum is of a light yellow-ochre colour and opalescent (physiological lipæmia, owing to a recent meal), and that the *direct* van den Bergh test is completely negative, even after an hour. The *indirect* test, however, shows the presence of bilirubin, estimated by the colorimetric method as eight van den Bergh units, that is to say, a bilirubin content of 1 in 25,000, or about twenty times the normal average. The indirect reaction in my case, it should be mentioned, takes place (April 30) remarkably slowly. In cases of ordinary obstructive jaundice a bilirubin-content of 1 in 50,000 in the blood-serum is sufficient to cause the presence of bilirubin in the urine. Dr. McNee also tested the resistance of the erythrocytes to hæmolysis in this case by adding the washed erythrocytes to graduated hypotonic sodium chloride solutions. He found that hæmolysis commenced with a solution of 0.48 per cent. (in a normal man taken as control it commenced with a solution of 0.50 per cent.), so that there was evidently no diminished resistance to hæmolysis as tested by this method.

It seems clear that the bilirubin in the blood-serum of ordinary cases of jaundice (in which it passes into the urine when it reaches 1 in 50,000 parts in the blood-serum) is chemically not exactly the same as the bilirubin in the blood-serum of this case and in the blood-serum of cases of congenital

<sup>1</sup> F. Parkes Weber, *Brit. Journ. Derm. and Syph.*, Lond., 1921, xxxiii, pp. 103-107.



or acquired hæmolytic jaundice. In this connexion Dr. McNee has kindly allowed me to refer to a very interesting case, that of a healthy young man with traumatic hæmorrhagic effusion into the knee-joint. On the eighth day after the injury Dr. McNee was able to examine some of the sanguineous effusion obtained by aspiration from the knee-joint. He found that it gave no direct but an *indirect* Hijman's van den Bergh reaction for bilirubin, equivalent to twelve van den Bergh units (that is, 1 in 16,666 parts of the fluid), and the reaction took place remarkably slowly, as in the blood-serum of my case (G. T. D.).

In conclusion I wish to thank Dr. O. May, to whom I was originally indebted for the case.

*Addendum.*—On a later occasion, when the sclerotics happened to be more deeply jaundiced (June 1, 1923), Dr. McNee very kindly again examined the blood-serum. He found then that it was clear (no meal had recently been taken) and gave a negative *direct*, but a positive *indirect* Hijmans van den Bergh reaction for bilirubin. The indirect reaction, which this time took place fairly promptly, was equivalent to eleven van den Bergh units (or 1 in 18,182). The blood-serum was free from bile acids, according to surface tension estimations by Mr. E. A. B. Pritchard, to whom my thanks are likewise due.

### Case of Erythræmia (Polycythæmia Vera, Vaquez-Osler's Disease), with Cerebral Hæmorrhage.

By J. A. RYLE, M.D.

PATIENT, a male, aged 48, admitted to Guy's Hospital, on January 18, 1923, had been under the care of Dr. J. A. Butler, and was transferred to Guy's Hospital for further observation. Dr. Butler reported red cells up to 12,000,000. His main complaint was that of "numbness of the left arm and leg, and also of the lips."

Family history: Unimportant.

Personal history: Pyorrhœa three years ago. "Gastritis" two years ago. At the beginning of November, 1922, patient suddenly experienced a sensation of numbness in the lips which extended down the left arm and leg and the hand and foot of the same side began to tingle. Attacks of this numbness were intermittent and came on every three or four hours. On attempting to walk patient found that he staggered slightly and felt as though he was "walking on rubber."

After a time he was compelled to give up work, as his gait was so much affected. Since the onset of these symptoms he has frequently suffered from headaches localized over the frontal region and at the same time his eyes have ached. At times his sight was impaired and objects about him appeared misty and blurred.

On admission: Patient represents all the features characteristic of erythræmia, e.g., excessively red facies, with a slight tinge of cyanosis of lips and finger-tips; extremely injected appearance of fauces and mucous membranes, suggesting acute inflammation; enlargement of the spleen; albuminuria ('06 per cent.); engorgement of the retinal veins, which are of a "blackberry" hue; and, in addition to these, the neurological symptoms referred to hereunder.

## BLOOD PICTURE.

Red cells ...	...	...	...	...	...	9,800,000
Hæmoglobin ...	...	...	...	...	...	110 per cent.
Colour index ...	...	...	...	...	...	0.5
White cells ...	...	...	...	...	...	18,750
Differential count: Polymorphs ...	...	...	...	...	...	64 per cent.
Large lymphocytes ...	...	...	...	...	...	10 per cent.
Small lymphocytes ...	...	...	...	...	...	21 per cent.
Eosinophils ...	...	...	...	...	...	4 per cent.
Coagulation time ...	...	...	...	...	...	Normal
Fragility ...	...	...	...	...	...	Normal
Mean diameter of red cells ...	...	...	...	...	Less than normal (as 6.4 to 7.2)	
Wassermann reaction ...	...	...	...	...	...	Negative

Radiograms of chest showed a normal heart shadow, increased striation and some patchy opacity in both lungs, but nothing to indicate enlargement or atheroma of pulmonary or other great vessels.

Nervous system: Patient walks with a distinct limp. There is a persistent numbness in the lips and left leg and arm with a sensation of tingling in the foot and hand which he describes as resembling "pins and needles." On one occasion he had a feeling of numbness in the left side of the nose which made it feel as if it was "running." Loss of tactile sensation was noted over areas of the left leg and arm and hand. Tendon reflexes slightly increased. Symptoms rather more pronounced after a bad headache.

*Note by Dr. C. P. Symonds.*—On rough examination of the visual fields there appears to be a homonymous defect in the left superior quadrants. I find sensory loss (left-sided) of a type which would correspond with a sub-thalamic lesion, together with slightly increased tendon jerks, abdominal reflexes much diminished as compared with the other side, and an indefinitely extensor plantar. This points to a lesion of the sensory pathway just below the right thalamus, cutting into the optic radiation. I take it this was a hæmorrhage.

Patient's progress in hospital was uneventful with the exception that he developed over the lower part of the abdomen and upper part of the right leg some tender reddish patches suggesting subcutaneous hæmorrhages.

A venesection produced slight temporary improvement in his subjective symptoms and a fall in the red-cell count to 7,000,000

Dr. PARKES WEBER said that the low colour index in this and many other cases of erythremia was of great interest. It might even be suggested that in such cases the low colour index of the erythrocytes was the primary defect and that the polycythæmia rubra was the expression of an automatic attempt on the part of the organism to compensate for a qualitative deficiency of the erythrocytes by an increase in their number. In the present case the average erythrocyte contained only about half the normal quantity of hæmoglobin, but the number of erythrocytes in the cubic millimetre of blood was about double the normal. Dr. Weber thought that the increase in the number of erythrocytes in many cases of *secondary* polycythæmia rubra was almost certainly of compensatory nature, and the low colour index in most cases of supposed *primary* polycythæmia rubra was an important fact that required further investigation by exact estimation of the hæmoglobin value in each case.

## Sections of Medicine and Ophthalmology.

JOINT MEETING.

Dr. G. NEWTON PITT, President of the Section of Medicine,  
in the Chair.

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### DISCUSSION ON "THE SIGNIFICANCE OF THE VASCULAR AND OTHER CHANGES IN THE RETINA IN ARTERIO-SCLEROSIS AND RENAL DISEASE."

Dr. G. NEWTON PITT (Chairman)

Said that this was a subject which interested both the ophthalmologist and the physician, and one on which the profession had yet much to learn. He hoped some new facts would emerge from this discussion.

There were one or two in particular upon which he would like information. One was, what was the relation between the amount of blood urea and the development of these retinal changes? If any observations on a great number of cases had been made—which he thought was doubtful—he hoped the results would be placed before the meeting.

The other was as to the relationship of retinal changes to arterial pressure and to arterio-sclerosis. In very many cases one could demonstrate arterio-sclerotic changes in the retina when retinitis was not necessarily present; and there were well marked cases of albuminuric retinitis in which the arterio-sclerotic change was not necessarily present. There was also the retinitis due to toxæmia which occurred during pregnancy, yet the retinitis would clear up and get well as soon as the child was born.

These were some of the points on which, particularly, information was desired. Also to what extent was retinitis dependent upon toxæmia, upon arterio-sclerosis, and upon high arterial pressure respectively?

Dr. H. BATTY SHAW.

I need hardly say that the task of opening this discussion on the significance of the vascular and other changes in the retina in arterio-sclerosis and renal disease is an exceedingly interesting one, because the ensuing debate will give two branches of our profession an opportunity to consider together the difficulties both meet with in the consideration of the above subject.

My presence here is largely due to the fact that owing to the generosity of my medical colleagues at University College Hospital I have had an opportunity of studying the clinical aspects and the post-mortem findings in a number of cases in whom the heart was found to be hypertrophied at the necropsy.<sup>1</sup> I may say at once that when I had gathered together the facts connected with this inquiry, and tried to fit them in with the various theories which had been advanced in explanation of them, I became quite bewildered. The only way in which I could explain the facts was to jettison former explanations, and look about for others. To show you how necessary this step was, I have merely to tell you of the following occurrences: (a) Some of the cases observed died of uræmic symptoms, and yet the kidneys did not reveal the changes usually described in these organs in such cases; (b) albuminuria was present in varying degrees of intensity, or was absent, and yet the kidneys gave no uniform appearances which could lead one to say why they were responsible for the former, or latter condition of the urine; (c) changes were met with in the retinae which in no way appeared to represent regularly what form or what detail of structure the kidneys would reveal in those cases; (d) some of the patients who during life presented signs of grave disorganization of the brain, showed at post-mortem examination that the brain was, to the naked eye, normal in appearance, and that the blood-vessels were free from changes in the middle coat or in the intima.

One particular phenomenon present in the cases studied seemed to be directly associated with the cardiac hypertrophy which they all presented; this particular phenomenon—hyperpiesis—was remarkable in one feature, viz., that it was so variable; in the course of a few days or so it would fall from a maximum to a minimum, not far removed from the normal; moreover the converse would occur, and neither clinical study nor post-mortem inquiry was able to reveal why these curious fluctuations occurred. In common with yourselves I had been taught to believe that this particular phenomenon was caused by a condition of the arteries known as arterio-sclerosis, an expression which has been used to cover an extensive field of change, but for the purposes of the discussion to-day I propose to limit it to that change met with in the middle coat of the arteries—the origin of which caused so much interesting discussion years ago as to whether it was due to hypertrophy or sclerosis of the middle coat—and to the other change due to the proliferation of the cells of the intima. The obvious question arose, how could this physical sign—hyperpiesis—vary so extraordinarily, and so quickly, between minimal and maximal heights, and yet be due to such a stable condition as that which we have called arterio-sclerosis? It was obvious that there was no such dependence. I could only explain the variability of the hyperpiesis by invoking the presence of a variable amount of a poison in the blood which, besides producing many hitherto inadequately explained clinical conditions, could also explain the changes in the middle coat of the arteries, as well as those in the intima.

I found that the invocation of toxic agents present in the blood also enabled me to explain away the difficulty of correlating many hitherto accepted signs and symptoms of "renal" disease, when little or no actual renal disease was present. I know for a fact that many other observers, both physicians and ophthalmic surgeons, would also wish to turn their eyes away from the view that arterial change is responsible for so much of what interests us in this

<sup>1</sup> "Hyperpiesia and Hyperpiesis (Hypertension)," Oxford Medical Publications, 1922.

discussion, and would rather look upon the change in the arteries as being a mere result of a toxæmia, working behind the scenes, and they have suspected that part of this toxæmia is bacterial in origin, and thus have explained some of those terminal episodes which present themselves to us in the form of hæmorrhages, pleurisy, pericarditis, &c.

With regard to the nomenclature of kidney disease, we know also that a break-away has occurred from older doctrines; too much of the change in the kidneys seemed to be allocated to a local dyscrasia of these organs, and an effort has been made to show that the changes in the kidneys are themselves direct products of the toxæmia originating from bacterial action at a remote site, or actually in the kidneys, or to a toxæmia the origin of which is at present unknown to us, but is certainly present as judged by its effects. This tendency towards a study of the blood has already led to a greater certitude of diagnosis as to the condition of the kidneys than any clinical examination of the body, or chemical study of the urine—at any rate so far as protein extrusions are concerned. Moreover, claims have been put forward to the discovery that changes in the arteries are due to a toxæmia of bacterial origin. We have known for years that intimal change can be produced experimentally by the injection of bacterial toxins, and now it has been claimed that the changes in the middle coat are simply of the nature of a chronic inflammation. As a physician, I feel that the time has arrived when we may safely discard the older doctrine which makes arterio-sclerosis responsible for the signs and symptoms we are to discuss to-day, and by concentrating our efforts upon the toxic view and supporting it as probable, we may thereby stimulate research which shall give this newer, or rather, revived conception, the support of demonstration.

Turning to the studies carried out by ophthalmic surgeons, it is well known that despite the strong views held by them that it is possible to differentiate arterio-sclerotic retinitis from albuminuric retinitis, they find difficulties. They admit that there is something wrong in their deductions, and we are familiar with the fact that they would prefer to speak of renal retinitis or the retinitis of renal disease, rather than of albuminuric retinitis, for reasons approved by physicians; indeed, as a physician, I feel that they have relied upon my branch of our profession too much, and have been satisfied too readily sometimes to accept our clinical diagnosis of some vascular catastrophe which has ended in paralysis or death. Further, they have gone so far as to admit that an arterio-sclerotic retinitis may be succeeded or accompanied by the signs of renal retinitis, and would argue that this is due to the fact that the harmful effects of arterio-sclerotic changes have progressed and, by involving the kidney, have led to the superadded changes formerly considered attributable to kidney disorganization. Such explanation has been widely held in the past, but as already intimated, evidence has been found which points to the possibly minor part played by the kidneys in the production of so-called "renal" signs and symptoms.

Some ophthalmological experts are already prepared to concede that the changes in the retina in renal retinitis are toxic, and that the seat of origin of these blood-borne toxic agents is pre-renal: so that, for them, even renal retinitis is a misnomer, just as is its predecessor, albuminuric retinitis. There are other weak points in the deduction made by some ophthalmic observers, namely, some of the changes met with in arterio-sclerotic retinitis are due in part to thickening of the middle coat, hence the silver or copper-wire appearance of the retinal arteries; other objective signs are due to thickening of the intima,

hence the irregular lumina of the arteries as seen by the ophthalmoscope. Now both of these changes indicate additions to, not subtractions from, the strength of these vessels.

How is it that hæmorrhages occur in the retina in arterio-sclerotic retinitis? When the hæmorrhage passes off no change of the nature of a rupture of retinal artery or vein has been demonstrated. As a physician, I am inclined to wonder whether the hæmorrhages are not more likely to be capillary in origin, and to have a genesis similar to that met with in the petechiæ of malignant endocarditis and other infective disorders. It is true that we have not yet arrived at a clear demonstration that these petechial manifestations are due to ruptured capillaries; but there is a large amount of analogical evidence that they are capillary in site of origin, and that they are of infective origin. Why should their genesis be different from that of the petechiæ developed elsewhere? And how are the white patches met with in arterio-sclerotic retinitis explained? Where do they come from? Or are they formed from local elements? If so, what provokes their formation? Surely not arterio-sclerotic changes! Are they not likely to be provoked by some similarly noxious agent, also brought by the blood stream?

We cannot, however, disregard the fact that most painstaking studies over very long periods have been made by ophthalmic surgeons, and a steady progress has been observed from changes met with in the retinal arteries to the development of hæmorrhages, of white patches, and even of papillædema, all due to arterio-sclerosis! There can be no reason whatever to carp at these findings, except that they need not be charged primarily to the account of the vessels. Is it not possible that changes even in the arterial walls leading to the thickening of the middle coat, with or without proliferative changes in the intima, are all due to a poisonous condition of the blood? Indeed, as already stated, one recent study has led to the deduction that the changes in the middle coat are of a chronic inflammatory nature, implying that an infective agency is behind them all. We also know from experimental studies already referred to, that intimal changes can be produced by the injection of bacterial toxins, and the study of syphilis shows incontestably what havoc can be engendered in the arteries by spirochaetes.

It is said that renal retinitis is always bilateral, and yet works by ophthalmic surgeons reveal the observation that "renal" changes may be shown in one retina by unilateral papillædema, which conforms with the well-known observation that blood-poisons need not necessarily produce symmetrical changes—unilateral Argyll-Robertson pupil and single wrist-drop in lead-poisoning being other examples of this anomaly. Further, I have seen figured in one work of reference "renal" retinitis which had lasted six years, though it is stated that cases of "renal" retinitis seldom survive two years—in contradistinction to cases of arterio-sclerotic retinitis in which the patients may live a great number of years: the duration of life after the development of both forms of retinitis seems somewhat variable.

If we need further disquieting evidence as to the dependence upon vascular disease, of hæmorrhages into the retina and the development of white patches, we may reasonably put forward the observation of ophthalmic surgeons that retinal hæmorrhages and white patches have been noted in pernicious anæmia, in the "fast disappearing disease" chlorosis, and even in the secondary anæmias due to peptic ulcer or to malignant ulcer of the stomach—diseases in which arterio-sclerosis is not charged with being the immediate cause. We may well ask, also, what is the source in secondary



anæmias of the development of soft-edged white patches in the retina which are quite indistinguishable from the "cotton-wool patches" of renal retinitis? Assuredly the changes in the "renal" retinitis and in the secondary anæmias are due to noxious agencies brought by the blood. It seems to me that it is likely that the changes met with in arterio-sclerotic retinitis are not due to the vascular change, but that the hæmorrhages and white patches and the slight change in the disc differ only from those met with in "renal" retinitis in their being called into existence by the slower operation of blood poisons, or by a decreased local tissue-sensitiveness in the former case as compared with the latter, or by the smaller amount of such poisons arriving at the scene of action in the former case, as compared with the latter. Some such explanation would also account for the longer survival of the former cases compared with the latter. It is taught that œdema of the limbs and body is a late arrival in the cases under consideration: possibly that is the reason why the white patches of arterio-sclerosis are so sharply defined and somewhat less extensive and are so "woolly" and so voluminous in so-called renal retinitis.<sup>1</sup>

If further evidence is needed that changes in the retina can be, and are, produced by a disordered blood-state rather than by changes in the kidney or by changes in the retinal vessels, we have it beautifully illustrated in eclampsia, where changes may be met with in the eye and in no other organ, such changes being remedied by abortion or by other evacuation of the womb.

Arterio-sclerosis has had a long innings as a cause of retinal change, but I submit that it is more reasonable to look upon arterio-sclerosis as a first effect, and arterio-sclerotic retinitis as a later one, of a toxæmia which acts slowly and in minimal quantity: when the toxæmia acts quickly or in accumulated large bulk the other type of retinitis results, and this other type needs another qualification than "renal," for the changes in the kidneys may be minimal, or the changes may be extremely variable, or extreme changes may be found in the kidneys with little or no change in the arteries or in the retinae. I would suggest that for the terms "arterio-sclerotic" retinitis and "albuminuric" or "renal" retinitis, or the retinitis of renal disease, we should substitute the terms "chronic" and "acute," "late" and "early" or "minimal" and "maximal toxic" retinitis, leaving for the future the investigation of the nature of the toxin concerned, the laws by which its potency is regulated and the reason why the kidneys should be so variably involved in different cases.

#### MR. R. FOSTER MOORE.

I propose to bring forward this evening what evidence I can in support of the view that in some cases of arterio-sclerosis a distinctive form of retinitis is developed, which is due, I believe, to the local vascular disease in the retina. I need do no more than say that the term retinitis is applied in the sense in which it is used in nephritis or diabetes.

It may be stated at the outset that these cases have usually been confused with renal retinitis, and I believe the statement that has been made and repeated, that renal retinitis in the old conveys a prognosis which is less serious than in the young, is in part due to the inclusion of the cases under consideration.

<sup>1</sup> Sir J. Herbert Parsons, F.R.S., "Diseases of the Eye," 1918; R. Foster Moore, "Medical Ophthalmology," 1922.



An endeavour will be made to establish the three following propositions :—

(1) That the ophthalmoscopic appearances of the condition are in large measure distinctive as compared with renal retinitis, the chief condition from which they have to be identified.

(2) That the retinal exudates are developed as the result of the local vascular disease in the retina.

(3) That as regards length of life and manner of death, this ophthalmoscopic condition implies a prospect which is in sharp contrast with that conveyed by renal retinitis.

I have collected in the table recorded (pp. 11-14) forty-seven cases of retinitis which I have seen on several or many occasions, and which I have had under observation in most cases for a number of years.

#### PROPOSITION 1.

It will, I believe, be agreed, that in the majority of cases of general arterio-sclerosis, the retinal arteries share in the disease to such a degree, that the condition in them is recognizable on ophthalmoscopic examination. Thus, of forty-four consecutive cases admitted to the wards of St. Bartholomew's Hospital on account of a gross vascular cerebral lesion, thirty-one, i.e., 70 per cent., provided undoubted evidence of retinal vascular disease; and we may go further and say, that an estimate as to the degree to which the general arterial disease has attained can be arrived at, with considerable accuracy, by the evidence which the retinal vessels supply.

It does not come within the scope of the present communication to describe these appearances, but as the general disease progresses, the disease in the retinal vessels becomes more marked, until, in a certain proportion of cases, exudates are developed in the retinal tissues, which I believe are due to the thickening of the coats of the arteries and to the reduction in their lumina, leading to impaired circulation in the tissues. I have elsewhere given reason for thinking<sup>1</sup> that the local pressure in the retinal arteries in these cases is less than the normal, though the pressure in the large arteries is greatly raised.

#### *The Ophthalmoscopic Appearances.*

It is not suggested that the individual spots or small areas of exudate, the presence of which is taken to justify the term retinitis, are pathognomonic, or that spots which at any rate, ophthalmoscopically, are similar do not occur under other conditions, but it is suggested that the spots themselves are in some measure characteristic in appearance, arrangement, and in the changes they undergo, and that when they are present in combination with marked vascular disease an ophthalmoscopic picture is presented which is in large measure distinctive.

The exudate takes the form of small whitish dots, or spots, or small areas: they have hard edges, and there is no pigmentary disturbance nor evidence of oedema around them; a spot of the diameter of one of the main retinal veins would be a rather large one. They occur chiefly in the central regions and are seldom copious; they may take the form of a partial or complete star figure around the yellow spot; at times they seem evidently arranged in relation to the radicles of the veins. Occasionally larger areas or small plaques are seen, formed apparently by the coalescence of spots which previously were discrete.

<sup>1</sup> *Trans. Ophth. Soc. U.K.*, 1916, xxxvi, p. 319.

In some cases the exudate is so scanty and the evidence of vascular disease is so conspicuous, that the exudate is overlooked or ignored amongst the much grosser and more obvious changes. In most cases flame-shaped retinal hæmorrhages are present; they are incidental to the vascular disease and have but little distinctive value.

A very striking feature of this form of retinitis is the frequency with which it is unilateral; thus out of forty-five of the present cases it affected one eye only in twenty-eight instances, i.e., it was unilateral in 60·7 per cent.; evidence of disease of the vessels was always present in the other eye. In renal disease retinitis may occur in one eye before it is evident in the other, but it very seldom remains unilateral for any long period.

The exudate is slow to develop and slow to undergo change, but if an accurate plan is made, it is easy to satisfy oneself that individual spots disappear and leave behind no trace of their former presence, but fresh ones are usually simultaneously appearing, so that the general aspect of the ophthalmoscopic picture may be maintained over long periods. The longest periods of which I have notes are seen in Cases XXXII and XLIII, in which retinitis persisted for eight years and seven months, and seven years and nine months, respectively. In other cases the exudate may entirely disappear; this happened in eight of the present cases, viz., Cases XII, XXVIII, XXX, XXXI, XXXV, XXXVIII, XLI, and XLVII.

In two of the cases disappearance of the exudate followed thrombosis of the retinal artery and seemed to be dependent upon the thrombosis for its occurrence, for in Case XXVIII retinitis was at first present in each eye, thrombosis then occurred in the left retinal artery, and disappearance of the exudate followed in this eye whilst it persisted in the other. There is good evidence that an eye which has been the subject of serious fundus disease, such for instance as thrombosis of the central vein or artery, or previous retinitis, or even high myopia, is as a consequence protected against the occurrence of retinitis in renal disease, and it seems that in the above mentioned cases another aspect of this phenomenon is exemplified, for retinitis was at first present, and the occurrence of thrombosis of the retinal artery seemed to determine its disappearance.

The chief points in which the ophthalmoscopic appearances differ from those of renal retinitis consist in the character and distribution of the exudate and the changes it undergoes, the frequency with which it is unilateral, its association with severe retinal vascular disease, the absence of œdema of the retina so that retinal detachments seldom if ever occur, and the absence of cotton-wool patches.

Finally, in this connexion, I should like to quote the following from the late Marcus Gunn's original paper on the ophthalmoscopic evidences of arterio-sclerosis,<sup>1</sup> in which he says:—

"In the most advanced cases the lines of the folds which radiate from the fovea centralis, due to the œdema, are sometimes eventually marked out by the deposit of white spots of degenerated effusion, so that we get the ophthalmoscopic appearances diagnostic of so-called albuminuric or renal retinitis, though in the variety now under consideration the condition may exist only in one eye and may not be accompanied by albuminuria."

This appears to be a good account of the condition we are considering: it is an account to which I had paid no attention previous to preparing this

<sup>1</sup> *Trans. Ophth. Soc. U. K.*, 1898, xviii, p. 361.

address. Amongst Gunn's fourteen cases there are six to which the above description would apply, and one may anticipate by saying that each of these six suffered from a cerebral hæmorrhage.

#### *Histology.*

These cases were followed as "out-patients," and consequently I have once only obtained a specimen for examination: I hesitate therefore to say much with respect to the histological characters of the exudate. In this specimen the spots were composed of small spherical areas of structureless hyaline material in the external molecular layer; they were deeply stained by orcein and took on a mauve colour in eosin and hæmatoxylin sections. So far as their structure goes they seem to be similar to the exudate which forms the "star figure" in renal retinitis, they are however smaller, there is no histological evidence of œdema, and no fat-containing phagocytic cells were present, such as may be seen in renal cases.

#### PROPOSITION 2.

That the retinal exudates are developed as the result of the local vascular disease in the retina and indicate a further advanced stage of it. This point can be considered in individual cases, and by a comparison of groups of cases. It is sometimes possible to watch, in the individual, exudate becoming super-added in the course of some years, where at first vascular disease alone was present. Cases XXVI, XXXII, XXXIV, XXXVIII, XL, XLI, and XLIV are examples of this.

Case XXXII is worth quoting in a little detail. The patient was first under the care of Marcus Gunn in 1908, at which time she had extensive arterio-sclerosis but no retinitis, her urine was free of albumin and sugar, and this was also true in 1909 and 1910. In 1911 she came under the care of the late George Coats, and then for the first time was found to have what he described as "white glistening spots disposed radially round the macula," and albumin in the urine. I saw her first in 1913 when she had a blood-pressure of 250 mm., a cloud of albumin in the urine, and retinitis in each eye; she was under my constant observation from this time till September 1, 1919, the date on which I last saw her. She had had a stroke in May, 1918, her blood-pressure was 260 mm., and she still had retinitis in each eye: thus for the first three years during which she was under observation she had retinal vascular disease only; she then developed retinitis, and this persisted more or less unchanged for eight years and seven months.

Again one may make a comparison between a group of patients in whom vascular disease alone is present in the retina, and another group in whom retinitis is present in addition. If, as I believe, the presence of exudates in the retina implies a stage of arterio-sclerosis in advance of a case in which vascular disease alone is evident, and if, as I am sure is the case, the disease of the retinal vessels increases *pari passu* with the general arterial disease, then a comparison of two groups of patients—in one of which vascular disease alone is present, and in the other of which exudates are present in addition to the vascular disease—should provide evidence that the latter group is composed of patients suffering from a further advanced stage of the disease. For this comparison I have available thirty-five patients belonging to the former group, and thirty-one belonging to the latter; the average age of each group is 59.

The average systolic blood-pressure of those without retinitis was 211 mm., and of those with retinitis 222 mm.

In March, 1919, seventeen (i.e., 48 per cent.) of the former group were known to have died, and eight were known to be alive, whilst on the same date twenty-one (i.e., 67 per cent.) of the latter group were known to have died, and six were known to be alive; further, on the same date, eleven of the first group were known to have suffered from a gross vascular cerebral lesion, and in eleven there was evidence that such had not occurred, whilst of the second group thirteen were known to have suffered from such a lesion, and nine had not done so.

It will be seen that, as regards the systolic blood-pressure, the incidence of death, and the frequency of gross vascular cerebral lesions, the second group manifests a higher grade of disease than the first. One may safely assert that these findings are at any rate compatible with the view that it is in the more advanced cases of arterio-sclerosis that retinitis is developed.

It is interesting to speculate, were it possible to examine the other tissues of the body during life under a magnification of fifteen diameters, as is done in the eye, whether changes in them would not be also found to occur, corresponding with these changes which are visible in the retina alone of the whole body, and especially would one expect such changes to be found in the brain tissues, for the retina is but a specialized part of the brain which is rendered subject to our examination.

### PROPOSITION 3.

Lastly, we come to the consideration of the length of life and the manner of death of patients who are the subject of this form of retinitis. It is undoubtedly true that few patients live so long as two years after the discovery of renal retinitis; thus Belt<sup>1</sup> found that of 419 patients, 94 per cent. died within two years, and Miles Miley<sup>2</sup> found in forty-five cases that the average duration of life after the discovery of retinitis was four months, and many other figures of a similar nature are available. We shall see that the group of patients we are now considering contrasts sharply with the foregoing.

As regards the prospect of life, the prognosis is of course somewhat grave; it is, however, less grave and more uncertain than in renal cases; such a patient may at any time develop a cerebral apoplexy, but, on the other hand, he may live for several or many years; thus, of twenty-eight patients who were known to have died, the average length of life after the discovery of the retinitis was two years and eight months,<sup>3</sup> whereas, as stated above, the average length of survival of the renal cases was four months only. Again, fifteen patients out of the twenty-eight, i.e., 53 per cent., lived for more than two years, whereas 6 per cent. only of Belt's renal cases lived for a similar period.

Whilst the prospect of life in these cases is very uncertain it will be seen how much better it is on the whole than in renal cases.

<sup>1</sup> *Journ. Amer. Med. Assoc.*, 1895, xxv, p. 735.

<sup>2</sup> *Trans. Ophth. Soc. U.K.*, 1888, viii, p. 134.

<sup>3</sup> Or, if one takes living and fatal cases together, the average length of survival after the discovery of retinitis is *greater than three years and 6.6 months*; I say "greater than," for it is clear, that as the "at present surviving" cases die, the average length of survival of the group will be increased.

The second point under this head refers to the manner of death of these patients. I have no precise figures, nor have I been able to find any, as to the proportion of patients with renal retinitis who die in uræmia, but it is certainly a considerable number. Of the present cases there are three only in which either nephritis or uræmia is given as the cause of death. I have received a report as to the cause of death in twenty-eight out of the thirty patients who are known to have died. Of these twenty-eight, fourteen, i.e., exactly 50 per cent., are known to have died of a gross vascular cerebral lesion, and in addition, four of the seventeen who were alive when last heard of, were known to have developed such a lesion which had not culminated fatally. I do not think it can be doubted that the vascular lesion was the dominant lesion in these patients, and that whilst no doubt the kidneys shared in the general disease, their function was not sufficiently impaired by it to threaten life seriously.

#### *Condition of the Urine.*

Having regard to the frequency with which albuminuria is intermittent in this class of patient, and seeing that most of them were watched as out-patients, the most that can be said is, that in seventeen cases the urine was albumin-free on one or more occasions. In some cases six or eight examinations were made, but in others the urine was examined once only, so that, no doubt, had the examinations been more frequent, the number of cases in which albumin was present intermittently would have been increased.

In conclusion I suggest that:—

- (1) In a proportion of cases of general arterio-sclerosis, as the disease of the retinal vessels increases, exudates form in the retinal tissues which are probably dependent upon the local vascular disease.
- (2) That the ophthalmoscopic appearances resulting are in considerable measure distinctive.
- (3) That the prognosis implied by this form of retinitis as to length of life, is quite uncertain, but may extend to several or even many years, and that it differs greatly from renal retinitis in this respect.
- (4) That a large number of these patients die of a gross vascular cerebral lesion, according to the present investigation 50 per cent., and
- (5) That the condition calls for separate recognition, and that the term "arterio-sclerotic retinitis" seems appropriate.

In explanation of the table of cases now following, in the second column is given the age of the patient when he or she first came under observation. The third column gives the systolic blood-pressure and is in most cases the average of several readings taken at different visits. The fourth column includes some details with regard to the retinitis. The fifth column gives the ultimate history so far as it is known, the evidence with regard to the occurrence of cerebral vascular lesions, and the cause of death where this is known. Cases I to XXX are those of patients who are known to have died, and Cases XXXI to XLVII are those of patients who were alive when the last information with regard to them was obtained.

TABLE OF CASES.

	Age	B.-P.	Details of retinitis	Ultimate history
I J. B.	45	245	June, 1913, retinitis right eye only; left arteries thin threads, no exudate	Died November 11, 1913, five months after the discovery of retinitis, of "cerebral hemorrhage"
II K. G.	49	215	January 30, 1914, star figure in right eye only; March 25, 1914, star still present	Died November 6, 1914, ten months after the discovery of retinitis, of a "cerebellar cyst"; post mortem
III A. C.	56	243	February 13, 1913, retinitis both eyes; November 27, 1913, retinitis increased, star figure left eye; January 15, 1914, retinitis still present	April 4, 1914, "three slight attacks of temporary loss of power of right arm and leg;" died November, 1915, two years and nine months after the discovery of retinitis; "a day or two before her death, which occurred suddenly, she had a hemiplegia; cerebral hemorrhage"
IV M. A. W.	68	255	November 4, 1913, retinitis right eye	"One year ago awoke and found three fingers of the right hand were funny and numbed, she has not recovered full use of them"; died November 14, 1913, ten days after the discovery of the retinitis, of a "paralytic stroke"
V A. C. W.	71	230	July 14, 1913, retinitis right eye	"Loses the use of her legs"; died May 29, 1916, two years and ten months after the discovery of the retinitis, of "interstitial nephritis and uremia"
VI A. C.	74	173	December 31, 1913, retinitis right eye	Died May 7, 1914, four months after the discovery of retinitis, "paralysed in speech and unconscious"
VII H. C.	54	220	December 14, 1912, retinitis both eyes; November 8, 1914, retinitis almost gone from both eyes with partial atrophy following oedema of the nerve	Died December 6, 1915, three years after the discovery of retinitis, of "cerebral congestion with symptoms of effusion"
VIII G. H.	60	265	October 20, 1913, retinitis both eyes	Died August, 1914, ten months after the discovery of retinitis, of "cardiac failure"
IX I. J.	50	290	January, 1913, retinitis left eye, with a star figure	June, 1913, "in Soho Square Hospital with a paralytic seizure"; died January 2, 1915, one year and eleven months after the discovery of retinitis, of "cerebral thrombosis"
X J. L.	53	210	February 3, 1914, retinitis both eyes, exudate along the radicles of the veins	Died February, 1915, one year after the discovery of retinitis, of "cardiac failure; a complication of diseases"
XI J. P.	53	205	April 30, 1913, retinitis with a star figure, left eye	Died October 4, 1913, five months after the discovery of the retinitis; "had a stroke, lost all power of his left side, died within twenty-four hours"
XII J. G.	63	235	March 6, 1913, retinitis, with a star figure beginning both eyes; December 18, 1913, thrombosis of artery has occurred in right eye with disappearance of exudate; left eye as before	Died April 14, 1914, one year and one month after the discovery of the retinitis, "in a fit"
XIII A. S.	63	—	March 4, 1915, partial star figure in right eye	Died September, 1915, six months after the discovery of the retinitis, of a "malignant growth of the liver"
XIV W. H. T.	66	—	October 25, 1913, retinitis right eye; February 22, 1914, still present	Died November 21, 1915, two years and one month after the discovery of the retinitis, of "chronic interstitial nephritis and uremia"
XV G. H. Q.	67	240	February 26, 1913, retinitis right eye; September 20, 1913, still present; March 7, 1914, still present	Died February, 1915, two years after the discovery of the retinitis, "in his sleep; cardiac failure"

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TABLE OF CASES—(Continued).

	Age	B.-P.	Details of retinitis	Ultimate history
XVI O. R.	71	225	February 1, 1911, retinitis both eyes; January 3, 1914, still present; February 7, 1914, still present	Died January 16, 1916, five years after the discovery of the retinitis, of symptoms of "cancer of the liver"
XVII F. B.	65	240	October 21, 1913, retinitis right eye	Died November 23, 1913, one week after the discovery of the retinitis, of "cerebral hemorrhage"; post mortem
XVIII E. C.	53	242	April 14, 1913, retinitis both eyes; March 12, 1914, retinitis reduced, both eyes	"Three years ago sudden attack of numbness running up both legs, went off after three minutes"; died February, 1917, three years and nine months after the discovery of the retinitis, of "symptoms of consumption"
XIX E. E.	55	—	March 13, 1914, retinitis both eyes, with a partial star figure; February 24, 1915, as before	Died October 14, 1915, one year and seven months after the discovery of the retinitis, of symptoms of "chronic renal disease and apoplexy"
XX A. A.	56	235	September 16, 1913, retinitis both eyes; March 3, 1914, still retinitis, both eyes	"Numbness in legs and thighs as if half dead"; died August 29, 1916, two years and eleven months after the discovery of the retinitis, of symptoms of "chronic nephritis"
XXI J. B.	65	300	June, 1913, retinitis left eye; November 4, 1914, still present	February, 1914, "had a stroke from which she has a good deal recovered"; died September 30, 1917, four years and three months after the discovery of the retinitis, "in a stroke, totally blind"
XXII J. G.	60	185	November 11, 1913, retinitis affected eye; March 7, 1914, still present	Thrombosis of central retinal vein; died March 4, 1918, four years after the onset of retinitis, of "consumption"
XXIII L. F.	52	237	June 12, 1912, retinitis right eye	Thrombosis of a tributary of the retinal vein; died April, 1920, seven years and ten months after the discovery of the retinitis, "of a stroke of which she had three"
XXIV A. H.	50	285	March, 1913, retinitis right eye; April 23, 1914, still present, a partial star figure	Died January 15, 1921, seven years and nine months after the discovery of the retinitis, of "diabetes mellitus"
XXV E. N.	55	—	—	Died nine years and six months after the discovery of retinitis, of "angina pectoris, no stroke"
XXVI C. S.	50	260	October 28, 1913, no retinitis; February 24, 1914, retinitis, right eye	Died April 5, 1914, ? cause; no evidence of a stroke
XXVII E. F.	68	225	Retinitis in eye without thrombosis only	Thrombosis of a tributary of the right retinal vein; died, date unknown, "suffered from cerebral hemorrhage"
XXVIII W. T.	63	220	December 30, 1912, retinitis both eyes; April 2, 1914 (when last examined) thrombosis of left central artery occurred and all exudate disappeared, still present in right eye, in which thrombosis did not occur	April 2, 1914, "no stroke, can walk three or four miles at a good pace"; died January, 1916, three years and one month after the discovery of retinitis, cause not known
XXIX A. S.	47	240	August 7, 1913, retinitis both eyes	1911, "stroke, right arm and leg useless, speech unintelligible"; died December 24, 1915, one year and four months after the discovery of the retinitis, of "Bright's disease, end came quite suddenly"
XXX W. C.	48	194	March 13, 1914, retinitis both eyes; March 12, 1916, retinitis disappeared	March 12, 1916, "slight paralytic stroke all down right side one year ago, speech affected, laid up four months, right side of face drawn"; died January 7, 1918 (? 1917), three years and ten months after the discovery of the retinitis, "after another stroke within twenty-four hours"



CASES WHICH WERE ALIVE WHEN THE LAST INFORMATION WAS ACQUIRED WITH REGARD TO THEM.

	Age	B.-P.	Details of retinitis	Ultimate history
XXXI A. D.	47	180	May 24, 1913, retinitis right eye only, well-marked star figure; November 15, 1913, star figure much less conspicuous; February 14, 1914, star figure gone except for one or two minute dots; November 17, 1914, star gone completely; February 13, 1919, no star figure and no exudate; thrombosis has occurred in the left central retinal artery	October 18, 1913, "suddenly taken with shaking all down the right side, right foot seemed to drag and have no use in it, all right leg numbed"; November 17, 1915, "twitchings all down right side"; July 18, 1918, "acute right hemiplegia and aphasia, blood-pressure 193 mm., urine no albumin"; February 13, 1919, "has been in bed ever since 1918"
XXXII A. H.	48	215	November 28, 1908; "extensive arterio-sclerosis; no albumin" (Marcus Gunn); January 28, 1911, "gross vascular changes, white glistening spots disposed radially round the macula" (George Coats); September 1, 1919, hæmorrhages and exudate still present in each eye (see text for fuller reference, p. 8)	November 28, 1908, first seen by Marcus Gunn who noted extensive arterio-sclerosis, no albumin; January 28, 1911, first seen by George Coats who found retinitis and albuminuria; since this time she has been under my constant observation till September 1, 1919; May, 1918, "stroke left side of face, left hand, and left leg;" September 1, 1919, "still gets about, blood-pressure now 260 mm., hæmorrhages and exudate still present in each eye"; retinitis is known to have been present for eight years and seven months. (See text for a fuller account, p. 8)
XXXIII L. S.	65	197	May 30, 1913, retinitis right eye	Under observation for five years and six months, gives a good account of a mild stroke in 1910, and of a second in 1911; June 4, 1916, "no fresh stroke"; February 12, 1919, "too feeble to walk, eyes about the same"
XXXIV M. M.	60	160	June, 1913, no retinitis; August 29, 1913, star figure just beginning in the left eye; September 16, 1913, star figure increasing	March 15, 1916, "my health is better, my eye does not trouble me"; retinitis known to have been present for two years and six months
XXXV E. P.	60	223	February 14, 1913, scanty exudate which completely disappeared	February 28, 1916, "thoroughly fit in every way"; retinitis present three years before last seen
XXXVI E. B.	67	210	October 6, 1913, retinitis right eye; April 18, 1914, still present	March 5, 1916, "am very well in health and my eye improves"; January 15, 1919, "general health very good, never any stroke"; retinitis known to have been present five years and seven months before last report
XXXVII A. B.	66	175	May 20, 1913, retinitis right eye; September 2, 1913; retinitis increased	March 5, 1916, "still active and able to take walking exercise and can read"; January 29, 1919, "father in very good health and leads quite an active life, remarkably active considering his age, 71"; "no stroke"; retinitis present five years and nine months before last report
XXXVIII C. L.	55	175	February 4, 1911, no retinitis; April 5, 1913, retinitis both eyes; October 25, 1913, retinitis still in right eye; disappeared from left	April 4, 1914, "no stroke, is in very good health"; retinitis known to be present one year before last report
XXXIX S. R.	65	205	Retinitis, both eyes	Not traced; June 30, 1916, "death certificate has not been issued" (Registrar-General)

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TABLE—(Continued).

	Age	B.-P.	Details of retinitis	Ultimate history
XL M. R.	62	137	August, 1908, no retinitis; August 31, 1921, retinitis both eyes	Thrombosis of venous tributary, right eye; under observation thirteen years, developed retinitis whilst under observation; August 31, 1921, "no stroke, walks to hospital"
XLI A. B.	49	195	May 16, 1913, no retinitis; March 25, 1914, retinitis in affected eye forming a star figure; September 10, 1921; no retinitis	Thrombosis of central retinal vein; September 21, 1921, "perfectly fit, nerves a bit shaky at times, no stroke": under observation eight years and four months; no retinitis present seven years and six months after its first appearance
XLII E. G.	—	—	April 13, 1909, retinitis in affected eye only	Thrombosis of central retinal vein: January 13, 1914, "quite lost her sight and been ill many months, unable to leave the house since last January"; retinitis present four years and nine months before last report
XLIII R. W.	54	155	December 16, 1913, retinitis affected eye only; August 22, 1921, very little retinitis still present	August 22, 1921, perfectly fit, no stroke, looks very well, blood-pressure 155 mm.; retinitis known to have been present for seven years and nine months
XLIV R. H.	66	245	March 23, 1913, no retinitis; February 2, 1919, retinitis, right eye	February 2, 1921, no stroke at any time, walks to hospital, blood-pressure 220 mm.; under observation five years and ten months; retinitis seen on last visit only
XLV G. H.	66	165	April 4, 1911, retinitis both eyes	January 15, 1919, "general health very good, never any stroke"; retinitis present seven years and nine months before last seen
XLVI J. B.	50	220	June 12, 1913, retinitis in affected eye only	Thrombosis of tributary of retinal vein; nothing further known
XLVII C. P.	56	222	March 7, 1914, retinitis right eye; March 24, 1916; retinitis disappeared; January 18, 1919, no retinitis	January 18, 1919, a year ago awoke suddenly with a giddy feeling which has never gone off, blood-pressure 210 mm.; retinitis present four years and ten months before last seen

## ANALYSIS OF CASES IN THE FOREGOING TABLE.

Forty-seven cases in all. Thirty of these are known to have ended fatally, and the cause of death is known in twenty-eight of them. Seventeen were alive when the latest information with regard to them was obtained, and of these there was satisfactory evidence that four had suffered from a gross vascular cerebral lesion.

*Prognosis as Regards Life.*—The average duration of life after the discovery of retinitis in the twenty-eight fatal cases was two years and eight months, and fifteen of them (i.e., 53 per cent.) lived for more than two years. The average duration of life of thirteen cases who were alive when the last information with regard to them was obtained was five years and five months. If one takes the forty-one cases, fatal and otherwise, in which the length of survival after the discovery of the retinitis is known, it works out at *three years and 6·6 months*.

*Cause of Death.*—Of twenty-eight fatal cases where the cause of death is known, fourteen, i.e., 50 per cent., are known to have died of a gross vascular cerebral lesion. In three cases only is nephritis or uræmia given as the cause of death. Of seventeen cases who were alive when the last information with regard to them was obtained, four were known to have suffered from a gross vascular cerebral lesion.

*Retinitis.*—Of forty-five cases the retinitis was unilateral in twenty-eight (i.e., 60·7 per cent.). In Cases XII, XXVIII, XXX, XXXI, XXXV,

XXXVIII, XLI, and XLVII, the retinitis disappeared whilst under observation.

*Urine.*—In seventeen cases at least the urine was intermittently free of albumin.

MR. PERCY BARDSLEY (Salisbury) :

I have long held the views of Dr. Batty Shaw on the toxæmic origin of retinitis and sclerosis.

Mr. Foster Moore kindly sent me a copy of his paper to study in advance. I agree with all that he has written in that paper, but, in the short time allowed me this evening, I must bring forward a somewhat different view.

While admitting that the picture of arterio-sclerotic retinitis, which he has so carefully and ably drawn, is correct in every detail, I think that picture only applies to cases of great chronicity. In other words, the retinitis depends on the acuteness of the disease producing the sclerosis. If the toxin is of a more drastic nature, or if exacerbation of the disease takes place, then the picture produced is indistinguishable from so-called renal retinitis.

Now the classical changes in so-called renal retinitis are four: (1) The retinal œdema, resulting in radiating lines or in a macular star; (2) the fatty spots; (3) the hæmorrhages; (4) the high pressure signs in the vessels.

These four cardinal signs may all be present in three groups of cases without albuminuria, viz.: (A) In intracranial pressure; (B) in advanced arterial sclerosis, perhaps I should say acute arterial sclerosis; (C) in many toxæmias. For instance, I myself have seen: (a) Several cases of syphilitic retinitis; (b) two cases of post-influenzal retinitis; (c) one case of unknown, but supposed cerebro-spinal origin; (d) also two monocular cases of pyorrhœic origin. In all these cases the four classical signs were present, and without albuminuria; yet they were indistinguishable from renal retinitis.

Since, then, these retinal signs occur so frequently without albuminuria, and since albuminuria can *only* be diagnosed by urinalysis, and not by the ophthalmoscope, I ask: "Is it not time that this misleading term 'renal retinitis' should be abolished?"

How, then, does the ophthalmoscope help us in these cases? It informs us that there is a toxæmia causing high blood-pressure and vascular inflammation with its accompanying sequelæ. The toxin may or may not at the same time be causing albuminuria; this is shown by urinalysis. The ophthalmoscope shows us also what is of the utmost importance, viz., whether this inflammation is accompanied or unaccompanied by sclerotic changes in the vessels walls.

This is of the *utmost importance*, for the prognosis of life or early death largely depends upon it.

If the ophthalmoscope shows advanced sclerosis, together with gross retinitis and albuminuria, then I believe the termination of life may be forecast in months, or even in weeks. If, however, the retinitis shows only high blood-pressure with *little* sclerosis, then the chance of recovery and fair length of life is good; the poison may be evacuated, the blood-pressure reduced, and no gross sclerotic changes left in the vessel walls. Of course, if the toxin is not acute enough to produce a retinitis, or only a mild form of retinitis, the prognosis is not so grave, even with advanced sclerosis. The sclerosis is the index of the chronicity of the poison, not of its acuteness. If you reject for the army the man with the hypertrophied heart because he is less able to

endure, you should also increase the premium of the life assurance policy of the man with hypertrophied arteries. That man will not withstand the toxin that produces retinitis as a man with normal arteries will.

Here I must challenge one statement made by Dr. Batty Shaw. He says: "Thickening of the middle coat produces the silver or copper-wire appearance of the retinal arteries." Now silver and copper-wire have very different sheens, and the respective sheen of each bears a very different interpretation. To-day I may see a patient whose arteries show neither. Next week, after a severe influenza, I see that patient with broad copper-bright light streaks and indented veins. The brachial blood-pressure reads 160 to 170. Have those arteries become sclerosed in two or three days? No! In a few weeks they may be back to normal.

Is it possible, then, to distinguish between the signs of simple high blood-pressure and the signs of arterio-sclerotic changes? I have stated in the past that this is possible, and, after five years' interval, I confidently reiterate that statement.

I would also emphasize a point that I think is not clearly grasped, namely, that one can detect sclerosis in the vessels when high blood-pressure is not present, and can thus forewarn the physician and the patient. Of this the following is rather a striking illustration:—

In 1911 a medical man sent his wife to me for refraction. Her age was 31. They had one child, aged 11.

After careful observation I wrote him that in my opinion it would be unwise to risk a further pregnancy, as her vessels showed considerable sclerosis. He was naturally very upset, and he took her to a well known physician, who found her blood-pressure a little under 130, and while he thought this perhaps a little high for her age, attached no significance to it.

A little later the lady became pregnant. At seven months albuminuria and eclampsia set in: the child was delivered, and died shortly after birth; the wife took more than a year to recover. She is still alive, and enjoying moderate health.

#### MR. PHILIP ADAMS (Oxford).

With reference to the so-called "renal" retinitis, this condition appears to be rare in the district in which I work. I hardly ever see a case nowadays, either at the Radcliffe Infirmary or the Eye Hospital, and yet I remember seeing it fairly often some years back. Only last week I was asked to examine the eyes of a man in the infirmary with chronic interstitial nephritis, but the condition present was one of arterio-sclerotic retinitis in one eye, thickened arteries and a hæmorrhage in the other. In contrast to this, towards the end of the war I was asked to see a man with a wound in the hip-joint, who was complaining of blurred vision; he had absolutely typical "albuminuric" or "renal" retinitis, slight papillædema, soft cotton-wool patches and hæmorrhages, with a stellate figure at each macula; but in spite of repeated examination of his urine nothing abnormal was found, except a very slight trace of albumin on the first occasion. His wound was very septic and draining badly, and he was extremely ill, but after amputation and free drainage he quickly recovered his health and sight, and he is alive and well at the present time. This case, shows, I think, that toxæmia can of itself cause the condition known as "renal" retinitis, without involvement of the kidney.

With regard to Mr. Foster Moore's propositions, I quite agree that there is

a distinctive condition of the fundus oculi, which he has named arterio-sclerotic retinitis, but I am inclined to think that the explanation of the condition given by Dr. Batty Shaw is likely to prove the correct one.

Again with regard to the length of time these patients live, my experience is similar to that of Mr. Foster Moore. Some years ago<sup>1</sup> I collected 159 cases of retinal vascular disease, exclusive of true "renal" retinitis, but including arterio-sclerotic retinitis and other retinal lesions associated with arterio-sclerosis, and I found that the patients in many of these cases lived to an advanced age provided their urine was free from albumin, whereas if albumin was present, eight or nine years was the maximum and this was quite the exception. Again, speaking generally, the older the patient at the time of onset of the eye symptoms, the less serious was the prognosis.

The more I ponder over my cases of vascular disease of the retina in arterio-sclerosis, the more convinced am I that one cannot make any prognosis on the eye condition alone; this must depend on the associated condition of the heart and kidneys. What is wanted, it seems to me, is, a comparison of the length of life between arterio-sclerotic patients who show no retinal change and those that do, because not all cases of arterio-sclerosis show distinguishing retinal changes, though the majority do so; and then one could form some idea of the true significance of these changes. This research could only be carried out conjointly by a physician and an ophthalmic surgeon working together.

#### DR. ARTHUR ELLIS.

At the London Hospital, Dr. Marraek and I are attempting to determine the relationship between disturbance of renal function and the occurrence of retinitis. With this end in view we are making a careful study of renal function in all patients in whom retinitis is determined. The tests of renal function employed consist in observations as to the presence or absence of albumin and casts, the determination of blood urea, the estimation of the power of excretion of phenol-sulphone-phthalein, the urea concentration test and observations on the occurrence or non-occurrence of fixation of the specific gravity of the urine. Up to date, twenty cases have been examined and the results obtained are shown in the following tables:—

Examination of these tables reveals two facts of major importance, first the constancy of high blood-pressure in these patients and second the possibility of separating them into two groups, one with gross disturbance of kidney function, the other without evidence of such gross disturbance.

(1) *High Blood-pressure.*—With one exception all the patients with retinitis examined showed high blood-pressure. In only three of the twenty was the systolic pressure less than 200: in one it was 180, in one 160, while in one patient the relatively low pressure of 148 mm. of mercury was found.

(2) *Differentiation of Cases according to Renal Function.*—In Table I are given those cases of retinitis in which gross disturbance of renal function was determined. It will be seen that in all nine there was marked urea retention, the figures for blood urea being in all these patients over 100 mg. per 100 c.c. There was also gross disturbance of phenol-sulphone-phthalein (P.S.P.) excretion, in six of the nine patients the excretion being either *nil* or only a trace. The urea-concentration test also, in every case in which it was carried out showed marked impairment of renal function, the ability to concentrate urea in the

<sup>1</sup> See *Brit. Journ. Ophth.*, 1917, i, p. 161.

TABLE I.—RETINITIS WITH GROSS DISTURBANCE OF RENAL FUNCTION.

Sex	Age	Duration of visual symptoms	Oedema	Blood-pressure	Albumin	Casts	Blood-urea	P.S.P.	Urea concentration test	Fixation of specific gravity	Remarks
F.	42	9 months	Slight	240/120	2.3	None seen	0.436	0	18 c.c. = 0.84 per cent.	No	Died, uræmia
F.	25	None	Slight	160/100	Cloud	None seen	0.400	0	"	Yes	Dying, uræmia
M.	38	2 months	None	210/160	1.5	Granular and hyaline	0.480	Trace	124 c.c. = 1.14	Yes	Died, uræmia
M.	46	1 year	Moderate	225/150	1.5	Granular	0.141	Trace	104 c.c. = 1.1	Yes	Died, broncho-pneumonia
M.	29	3 months	None	200/140	2.5	Granular and hyaline	0.107	Trace	"	Yes	Died, uræmia
F.	43	2 months	None	220/135	2.5	Granular and hyaline	0.141	Trace	"	Yes	Died, uræmia
F.	30	2 weeks	Slight	280	1.6	Granular	0.109	6 per cent.	80 c.c. = 0.87	No	"
M.	23	1 week	Slight	240/170	2.3	Granular and hyaline	0.171	7 per cent.	62 c.c. = 0.96	Yes	"
F.	25	3 months	Slight	210/160	1.5	Granular and hyaline	0.120	7 per cent.	58 c.c. = 1.25	Yes	"

Total number = 9. Average age = 33. Average B.P. = 220.

TABLE II.—RETINITIS WITHOUT GROSS DISTURBANCE OF RENAL FUNCTION.

Sex	Age	Duration of visual symptoms	Oedema	Blood-pressure	Albumin	Casts	Blood-urea	P.S.P. <sup>1</sup>	Urea concentration test	Fixation of specific gravity	Remarks
F.	54	2 years	Slight, both legs	280/170	1.3	Granular	0.080	15 per cent.	93 c.c. = 1.86 per cent.	No	Early cardiac failure
F.	36	1 month	None	180/120	Cloud	Hyaline	0.043	26	110 c.c. = 1.44	No	Onset in pregnancy
M.	56	3 months	None	250/160	Cloud	Granular and hyaline	0.045	23	"	No	Hemiplegia
F.	51	6 months	None	250/170	Trace	None seen	0.040	32	68 c.c. = 2.1	No	Died, "cardiac failure"
M.	54	"	Moderate (heart failure)	210/160	1.8	Many, hyaline	0.035	30	75 c.c. = 2.2	No	Died, cardiac failure
M.	37	3 months	None	250/160	1.10	Hyaline	0.030	30	95 c.c. = 2.3	No	Died, cerebral hemorrhage
F.	43	2 years	None	280/180	Cloud	Hyaline	0.020	48	60 c.c. = 2.28	No	"
M.	57	2 months	None	280	Trace	Granular and hyaline	0.048	49	"	No	"
F.	35	None	None	225/160	None	None seen	0.036	52	128 c.c. = 2.25	No	Hemiplegia, now marked loss of vision
F.	30	1 month	None	220/150	1/10	Granular and hyaline	0.026	55	76 c.c. = 1.98	No	Onset in pregnancy
M.	49	"	None	145/85	Trace	Hyaline	0.040	68	73 c.c. = 2.3	No	"

Total number = 11. Average age = 46. Average B.P. = 235.

<sup>1</sup> The phenol-sulphone-phthalein employed in these tests gives a low figure for the normal, 40.70 per cent., instead of the 60.90 per cent. given by that supplied by Hynson, Westcott and Dunning, on which most normal figures are based.



urine being in the neighbourhood of or under a concentration of 1 per cent., instead of the normal of over 2 per cent. In six of the nine patients there was fixation of the specific gravity of the urine. Four of these nine patients are known to have died of uræmia, one is dead of broncho-pneumonia and two others are dying of obvious renal inefficiency.

In Table II are given the findings in those cases of retinitis in which evidence of gross disturbance of renal function was lacking. There are eleven patients in this group. In only one was there any evidence of nitrogen retention: this patient had on admission 80 mgm. and two months later 60 mgm. of urea per 100 c.c. of blood. It will be noticed that in this patient the phenol-sulphone-phthalein figure was also the lowest for any patient in the group and the urea concentration also below normal. The patient was suffering from cardiac failure and it is probable that the poor figures for renal excretion were in part dependent on the circulatory failure. In the other ten patients the figure for blood urea was within normal limits. The phenol-sulphone-phthalein excretion was less than the normal in more than half the cases in the group, but with the exception of the patient showing urea retention it did not approach the condition found in cases of advanced renal disease.

The results of the urea-concentration test in this group of patients was particularly interesting. In only three was the figure below the normal 2 per cent. One of these three was the patient already mentioned with urea retention and low phenol-sulphone-phthalein, the other two were both cases of retinitis occurring in pregnancy—a retinitis recognized by the ophthalmologists as different on account of its much more favourable prognosis. None of the patients in this group showed fixation of the specific gravity of the urine. Of this second group of patients three only are known to be dead, one of cerebral hæmorrhage and two of cardiac failure. It is to be noted that two other patients in this group are hemiplegic.

We see then that patients with retinitis may be divided into two groups, one showing gross impairment of renal function, the other not. In the former death usually occurs relatively soon after the patient seeks admission to a hospital, the common termination being uræmia. In the latter, life is more prolonged but vascular accidents are frequent and are probably the common eventual cause of death.

With what are we dealing in these two groups? This opens up an interesting question. Are they the same disease, one being an advanced stage of the other, or are they two separate and distinct diseases? Is one primarily a renal disease with a secondary rise of blood-pressure, and the other primarily a high-pressure disease with renal involvement merely as a secondary result?

If the renal cases do represent the end stage of a disease the earlier manifestations of which are seen in the "vascular" group, then we should expect the patients listed in Table I to be older and to have higher blood-pressures than those in Table II. The reverse proves to be the case, the average age in the "renal" group being thirteen years younger than in the "vascular," and the average blood-pressure in the "vascular" group 15 mm. higher than in the "renal." This is in keeping with experience and in agreement with the generally accepted differentiation of two diseases, (a) chronic nephritis with high blood-pressure, and (b) essential vascular hypertension with secondary renal damage. Is there a different ophthalmoscopic picture in these two conditions? Will these ophthalmoscopic appearances coincide with Mr. Foster Moore's renal retinitis on the one hand and arteriosclerotic retinitis on the other? These are questions which require answering, and to-day's discussion should help to hasten their solution.



Dr. C. O. HAWTHORNE.

I propose to limit my remarks to the various descriptive or diagnostic terms that have been proposed in the debate: these terms have been suggested as appropriate when retinitis is associated with renal disease on the one hand, and, on the other, when retinitis is free from that association but exists in the presence of more or less conclusive evidence of arterial degeneration. In Dr. Batty Shaw's view the retinitis existing in these two sets of circumstances is one and the same: it is due to a toxic condition of the blood, and is produced by a direct action of the toxin on the retinal tissues. Hence Dr. Batty Shaw wishes to call the condition toxic retinitis. On the other hand, Mr. Foster Moore recognizes two forms of retinitis, though he admits he is not always able to distinguish the one from the other. One form Mr. Foster Moore regards as a result of advanced degenerative change in the retinal vessels, and he therefore calls it arterio-sclerotic retinitis; and the other he takes to be an expression of renal disease, and hence he applies the term renal retinitis.

I challenge both sets of proposals, not on the ground that the underlying propositions are not true, but on the ground that we do not know them to be true. Hence it follows that terms implying their truth ought not be admitted to a scientific vocabulary.

Each of these terms—toxic retinitis, renal retinitis, arterio-sclerotic retinitis—involves an undemonstrated speculation or hypothesis, and this consideration alone is sufficient to condemn such terms as bad and as inadmissible to a scientific nomenclature; for scientific terms, if pretending to be descriptive, should be descriptive of facts and not of opinions about facts. Already in medicine there are more than sufficient areas within which confusion is perpetuated and counsel darkened by words without knowledge; and any extension of this method should be resolutely resisted.

Dr. Batty Shaw knows nothing of his hypothetical toxin, except from what he judges to be its effects; he cannot tell what the toxin is, nor where it comes from, nor how it acts. It is one thing to teach or to argue in favour of a certain theoretical explanation, and quite another thing to impose this explanation as settled doctrine by incorporating it in a descriptive title.

Similarly, Mr. Foster Moore's terms are obviously speculative or hypothetical. In time they may turn out to be justified, but at present they are far in advance of the facts. There is the more reason for circumspection here, seeing that Mr. Foster Moore allows that the two pictures he draws are not in all cases confidently distinguishable the one from the other.

In a word, it may be urged that into clinical names and phrases we ought not to introduce terms implying a confident knowledge of causation, when, as a matter of fact, such knowledge is not in our possession.

Dr. J. F. GASKELL (Cambridge).

I will endeavour to give my views as shortly as possible on those forms of disease which bear upon the present discussion.

The point in this discussion I want to emphasize above all others is that two conditions of entirely different pathology are concerned: one being primarily a disease of the kidneys, the other of the vascular system as a whole. It is inevitable, owing to the close interdependence of these two systems, that lasting disease of one should affect the other, so that a composite picture is ultimately formed in which both systems are affected.

Dealing first with the condition which is primarily renal, I hold that the initial occurrence is an acute inflammation of the whole organ, in which every structure of which it is built up is involved—glomeruli, tubules, their supporting or interstitial tissues, and very probably the intrinsic blood-vessels of the organ also.

There are various stages of this diffuse nephritis with which are usually associated those forms of retinitis called albuminuric, or better, renal retinitis.

The great damage which the kidney thus undergoes calls for an increased efficiency in the circulatory system in order that the excretory function should be adequately carried on, and cardiac hypertrophy and increased blood-pressure result. The essential pathology is still however renal, and the one danger to be apprehended in the later stage, where such an hypertrophy has been successfully brought about, is renal failure in the form of uræmia.

The second condition is a cardio-vascular one in which three factors invariably take part—a raised blood-pressure, cardiac hypertrophy and disease of the small arterioles of certain organs of the body. Organs which are always affected are the brain and the kidney, around which the discussion turns to-night. It is to this class that I consider Mr. Foster Moore's series of cases belongs, and I am fully in agreement with him that they should be separated from any connexion with renal conditions. Another point that I want particularly to emphasize is that in the purest examples of this condition the vascular changes, which are degenerative in nature, are entirely confined to the small peripheral arterioles. The largest arteries are wholly free from any such change; on the contrary they frequently manifest a true muscular hypertrophy enabling them to cope with the increased pressure of the blood-stream.

In these cases the cause of death is almost always to be traced to the changes which occur in the brain, cerebral hæmorrhage being by far the commonest occurrence. There is, however, another group of events depending upon relative failure in the cerebral circulation, which also leads to death. The disease of the cerebral arterioles, especially in the medulla, becomes so great that a local cerebral anæmia is brought about if the high level of the blood-pressure is not maintained. Death often follows without gross cerebral lesion, but with signs of respiratory failure, such as Cheyne-Stokes breathing, &c. This condition must not be confused with uræmia.

With regard to the cause of this condition and the sequence of the three chief events, it is very difficult to form an opinion as to whether the arterio-sclerotic changes of the arterioles are secondary to the high blood-pressure, or whether the high blood-pressure is a response to obstruction to the peripheral circulation through essential organs. I do not myself consider that the invoking of a "toxæmia" without any evidence of its nature adds to the clearness of any view of the pathology of this condition. I am, however, in hearty agreement with the opener of the discussion that the changes are due to some cause which acts upon the circulation as a whole, and that the sooner terms such as chronic interstitial nephritis, which imply that the kidney is the primary cause, are given up, the better. The term I incline to adopt for the condition is one which we owe to Sir Clifford Allbutt, namely, "hyperpiæsis," which both draws attention to the circulatory system as the primary system involved, and emphasizes one, and perhaps the primary, of the factors which constitute the pathological entity.

(The Discussion was adjourned till December 8, 1922.)

## Sections of Medicine and Ophthalmology.

JOINT MEETING.

Chairman—Mr. A. L. WHITEHEAD, President of the Section of Ophthalmology.

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### ADJOURNED DISCUSSION ON "THE SIGNIFICANCE OF VASCULAR AND OTHER CHANGES IN THE RETINA IN ARTERIO-SCLEROSIS AND RENAL DISEASE."

Mr. ERNEST CLARKE.

I think we shall find that when their statements have been thoroughly confirmed the excellent papers of Dr. Batty Shaw and Mr. Foster Moore will be really "epoch-making." What many of us have been long suspecting has at length materialized. Those who can go back as many years as I can will remember that we were taught (and believed all this time until recently) that high blood-pressure was the *cause*, or one of the causes, of so-called "hæmorrhagic retinitis" in the majority of cases. We sent our patients to a physician for him to lower the blood-pressure and fortunately for the patient, even bleeding did not do this. Now we are beginning to see that high blood-pressure is *not* the cause, but *one of the symptoms* (we may even believe that it is a *protective measure*), and we must go further back for the cause which in most cases is some form of toxæmia. This being so, is not the term "hæmorrhagic retinitis" (which most of us disliked because of its unscientific style) probably the best term we can use at present as indicating the trouble and not binding us to any theory (and thus it would, I suppose, meet with the approval of Dr. Hawthorne).

Mr. Foster Moore, under "Proposition 2," makes this remark:—

"If, as I believe, the presence of exudates in the retina implies a stage of arterio-sclerosis in advance of a case in which vascular disease alone is evident, and if, as I am sure is the case, the disease of the retinal vessels increases *pari passu* with the general arterial disease, then a comparison of two groups of patients—in one of which vascular disease alone is present, and in the other of which exudates are present in addition to the vascular disease—should provide evidence that the latter group is composed of patients suffering from a further advanced stage of the disease."

This of course tallies with the statement made by Dr. Batty Shaw, who suggested that the different stages of disease are due to the amount of poison, and the time during which it is acting.

With reference to the so-called "exudates," why should they be present in one eye and not in the other? Is it due to the same cause which we think

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operates in unilateral hæmorrhage, namely, that the tension of the affected eye is lower than that of the other? It would be a useful investigation if, in the future when we find hæmorrhages or exudates, or both, in one eye only, we were to take the tension of both eyes with a tonometer.

We ophthalmic surgeons are quite familiar with retinal hæmorrhages occurring when the tension of the eye is lowered; it is an unfortunate accident which may occur in doing an iridectomy for glaucoma, or extracting a cataract, but we have always assumed in these cases that the vessels were diseased.

Dr. Batty Shaw has very rightly hinted that the thickening of the middle coat and of the intima strengthens the vessels, and we have sufficient evidence to show that this thickening or sclerosis is inflammatory in origin and is only followed by degenerative changes if the blood supply is deficient. Both the openers of this discussion have suggested that the hæmorrhages do not come from ruptured vessels. Dr. Batty Shaw suggests that a hæmorrhage may be of the nature of a leakage from the capillaries; this would seem to be confirmed certainly in those cases in which small hæmorrhages are unaccompanied by any retinitis, and which disappear without leaving any trace, and appear to be unassociated with any constitutional disease. Such cases, I take it, constitute an example of the first stage which, if allowed to continue, may develop into so-called "arterio-sclerotic retinitis," which itself, if allowed to continue, or if the toxins are increasing, may lead to renal disease, and so eventually to the final stage of "renal retinitis."

Dr. W. N. GOLDSCHMIDT.

The success of any attempt to divide up cases of retinitis into those due to renal "mischief" and those due to other disorders depends partly on the decision as to what symptoms and signs, apart from retinitis, justify the labelling of a case as "renal." The following record illustrates the difficulty of this problem.

It is of interest from the point of view of the relationship between the symptoms and signs of "parenchymatous nephritis" and the condition actually found in the kidneys, both during the disease and at the post-mortem examination. The effect of decapsulation and other remedies is also discussed.

On January 28, 1922, a man aged 42 was admitted to hospital complaining of swelling of his legs for seven weeks and of his "stomach" and back for six weeks. Additional symptoms were very troublesome, viz., flatulence, shortness of breath on exertion and occasional headaches. The only serious illness from which he had suffered was a febrile attack ("influenza") in Egypt in 1918. There was no venereal history.

Condition on examination: There were great pallor and marked œdema of legs, abdomen and back, and also ascites. The cardio-vascular system showed no abnormalities, though the heart sounds were very faintly heard. The systolic blood-pressure was 132 mm. In the respiratory system there was evidence of some pleural effusion at both bases, especially the right. The tongue was furred, the tonsils rather injected and the gums showed pyorrhœa. The abdomen was distended, not only by fluid but by meteorism as well, especially in its upper part. He complained of a dull pain all over his abdomen and great flatulence after meals. Urine: About 200 c.c. were passed in twenty-four hours, specific gravity 1030, brown in colour, acid, and containing about 2 per cent. of albumin: microscopic examination showed red blood cells and numerous granular, hyaline and epithelial casts: the urea-concentration by MacLean's test was nearly normal, averaging 1.7 per cent. The blood-urea was within normal limits (33 mg. per 100 c.c.). The Wassermann reaction was negative. The fundi, examined ten days after admission, were normal.

*Treatment* at first consisted of rest in bed, salt-free diet and a diuretic mixture containing potass. acetat., tr. scillæ, sp. aether. nit. and succ. scoparii. In four days the quantity of urine passed in twenty-four hours rose to 660 c.c. On the sixth day hot-air baths once daily were used, but no satisfactory perspiration was produced. The bowels were also very obstinate, and large doses of jalap, given with hot water, were required to achieve any result. On the tenth day, urea (1 dr.) was given four times a day for a few days, without any effect on diuresis, and then diuretin (20 gr. t.d.s.), but with no result: the urine continued to average about 500 c.c. daily. He was drinking fairly freely, but little fluid was excreted, either by the skin or by the bowels. The flatulence was very troublesome, and had to be relieved by hot applications and carminatives.

Since no progress was being made by the aid of medicinal and dietetic measures, and since the history was comparatively short, it was decided that the effect should be tried of decapsulating a kidney. Accordingly three weeks after admission his right kidney was dealt with in this way by Mr. Gwynne Williams. One kidney only (the right) was operated upon to begin with, in order to watch the result. The kidney was found to be of normal size and colour: a slit was made in the capsule (which was not tense) and a small piece of kidney substance was removed for examination: the capsule was then separated from the kidney all round. The peritoneal cavity was opened and 2 to 3 pints of ascitic fluid allowed to escape: this was milky and opalescent from the presence of lecithin-globulin (Dr. Mackenzie Wallis). The intestines were pale but there was no visible peritonitis.

Four days later the urine had risen to 900 c.c., and the œdema was rather less: ten days after the operation it rose suddenly to 1,200 c.c. (albumin, 0.36 per cent.), but within four days it dropped as quickly to 600 c.c. Urea was now again tried and pushed in doses of 15 grm. t.d.s. except when the patient was sick; the urine now increased within eighteen days to 1,600 c.c. in twenty-four hours and then averaged 1,200 c.c. for about twelve days. The general condition of the patient was, however, but little altered and the œdema not materially diminished. The abdomen and scrotum were again tense.

With the idea that the increased urinary output might have been due partly to the operation, it was decided to decapsulate the left kidney. Accordingly, on April 5, 1922, seven weeks after the first operation, a second operation was performed by Mr. Gwynne Williams. The kidney and its capsule were normal in appearance and size: the capsule, which was not tense, was slit and retracted: the peritoneal cavity was opened and several pints of milky fluid like that at the first operation were removed. The intestines were pale but otherwise normal. For the next two days the quantity of urine dropped to 700 c.c., albumin still being present. On the third day the urine rose again to 1,350 c.c., but after that it steadily decreased in amount and the œdema reaccumulated. Three weeks after the operation an attempt was made to reduce the œdema by giving him less to drink (only about 1½ pints daily), salt-free diet, purgatives and theocin sod. acet. 4 gr., tr. digitalis 7½ minims, and sod. sulph. 15 gr. t.d.s.: the increasing oliguria could not be stayed.

On April 27 the blood-urea had risen to 48 mg. in 100 c.c. The abdomen was now very distended again and vomiting started. On May 9 everything else having failed to relieve the œdema it was considered justifiable to insert Southey's tubes into his feet, which was done with scrupulous aseptic precautions. A very large quantity of fluid was drained off (4 pints on the first day). The abdominal distension, which was partly gaseous, was not relieved. The chlorides in the urine were distinctly diminished.

After a few days the feet became red and painful: the temperature dropped to 96 F.; the blood-pressure, which had been 132 mm. Hg on admission, soon dropping to 120 mm. where it remained, now fell to 90 mm., and he became drowsy and sometimes semi-comatose. On May 30 his blood-urea had risen to 63 mg. in 100 c.c. The albumin in his urine on June 1 was 0.5 per cent., on June 2, 1.25 per cent., and on June 3, 2.5 per cent. He was now somnolent, often sick, and there was evidence of cellulitis in the legs. He died on June 5.

*Autopsy.*

Externally a few petechiæ were seen on the arms. Incisions into the dorsa of the feet showed the cellular tissues to be distended with pus. Heart was small, only  $5\frac{1}{2}$  oz. in weight, and atrophic. The heart muscle was dark brown. A few patches of atheroma were seen in the aorta and coronary arteries. Lungs, &c.: Each pleural sac contained about 6 oz. of clear fluid: scattered fibrous adhesions on both sides. The lungs were congested and œdematous. Peritoneal cavity contained a large quantity of turbid ascitic fluid; considerable flakes of fibrinous exudate on the peritoneal surface. Intestines: Sigmoid colon contracted, apparently by thickening of bowel walls; mucous membrane showed slaty-grey patches, and was œdematous and congested; no ulceration or evident scarring; the affected area was sharply marked off from the contiguous normal gut.

Kidneys: The left kidney, normal in size, was removed and injected with preservative fluid soon after death. The right kidney, of normal size and weighing 5 oz., was not injected with preservative fluid; the surface was smooth and rather pale; the cortex and medulla were normal, and so were the vessels.

*Microscopic Sections of the Kidneys.*—(Described by Mr. T. W. P. Lawrence.)

"(1) *Section of right kidney, piece being removed at the first decapsulation operation:* (a) There is localized cloudy swelling and necrosis of the tubules, probably of an acute nature; adjacent areas are quite normal. (b) There are some slight chronic changes shown by thickening of the capsules. (c) There is proliferation of the epithelium of the glomerular tufts. (d) There is slight increase of the interstitial tissue between the tubules in places. (e) There are small localized effusions of red corpuscles.

(2) *Section of right kidney, piece being removed at the post-mortem examination, the organ not having been injected with preservative fluid:* (a) All acute changes above described are absent. (b) Chronic changes are shown by thickening of the capsule, only slight, as above. (c) The numbers of nuclei in the glomerular tufts are fairly normal. The corpuscles are not so large as in the above section. (d) Slight interstitial changes between the tubules, as in the above section. (e) No effusion of red corpuscles. (There is considerable autolytic change in the tubular epithelium.)

(3) *Section of left kidney, piece being removed at the post-mortem examination, the organ having been injected with preservative fluid very soon after death:* The microscopic details of this kidney were identical with those met with in the right kidney (*see* (2) above), no acute cloudy swelling or necrosis being visible in the tubular epithelium. The autolytic changes were minimal.

*Summary.*—The sections of the piece of kidney removed during life showed slight but definite changes, such as could be produced by some toxic agent arriving by the blood-stream. The sections of the pieces removed after death only showed the slight chronic changes met with in the sections of the piece removed during life, and were very little different in appearance from those of a normal kidney."

*Comment on the Case.*

This man died from an illness, the symptoms of which were typically those which have been attributed to "chronic parenchymatous nephritis"; but his kidneys were found at the post-mortem examination both to be normal macroscopically and practically normal microscopically. His actual death was perhaps due to or hastened by the infection which followed insertion of Southey's tubes, but he was steadily progressing to the inevitable end before the use of these was reluctantly resorted to. The section from the piece of right kidney taken during life at the first decapsulation operation showed some parenchymatous changes of an apparently acute nature. As the right kidney showed none of these appearances at the post-mortem examination, decap-



sulation may have restored the kidney, but did not improve the pre-renal toxæmia.

I am forced to the conclusion (unless in such cases one thrusts aside as valueless *all* microscopical evidence of the presence or absence of disease) that this long, insidious and fatal illness was not accounted for primarily by the state of his kidneys. The heart was very small and in a condition of brown atrophy; this organ, therefore, though the condition of brown atrophy is always looked upon as a secondary phenomenon and not a primary morbus cordis *sui generis*, actually showed considerably more evidence of disease than the kidneys. Even supposing the kidneys *had* shown parenchymatous changes at the post-mortem examination, there would be no more reason for considering the kidney degeneration primary than that of the heart. Incidentally, it must be remembered that there were no signs of renal inefficiency during the earlier and greater part of his illness, except oliguria (which occurs in heart disease), albuminuria (which occurs in 6 per cent. of healthy adults), and diminished excretion of chlorides (which occurs also in fevers, especially pneumonia, and is not incontestably the result of damaged kidneys). Furthermore, the œdema is not easy to explain as due entirely to the retention of chlorides, since œdema is not a feature of pneumonia, and does not even occur in obstructive suppression of urine or after removal of the only kidney. It is of great importance to note that, although towards the end of his illness the kidneys were only slightly damaged or possibly not at all, the blood-urea rose, so that an abnormally large amount of urea may be present in the blood with but slight or no changes in the kidneys.

It would, therefore, seem as if this man's illness were due to some pre-renal poison which caused exudation into the tissues, and incidentally damaged some of the tubules of the right kidney, but these latter had recovered by the time the man died.

I wish to thank Dr. Batty Shaw and Mr. Gwynne Williams for kindly allowing me to make use of their case reports, and Mr. Lawrence for his description of the microscopical sections.

#### Mr. D. LEIGHTON DAVIES (Cardiff).

I suppose the majority of ophthalmic surgeons will agree that there is a distinct clinical difference between a hæmorrhagic retinitis due to, or associated with, marked arterio-sclerosis on the one hand, and a retinitis due to or associated with albuminuria on the other. On the one hand we see a fundus in which the disc has a deep brick-red colour, with round, irregular or flame-shaped hæmorrhages scattered about the retina, but mostly somewhere in the neighbourhood of the blood-vessels; (it may be also that near these same vessels a few small white spots can be seen); arteries glistening and rather tortuous, and perhaps uneven in calibre, whilst the veins are full. Such is the clinical picture of a retinitis associated with arterio-sclerosis.

On the other hand we have a fundus, of which the disc is perhaps of a pink colour and the edges tend to be a little feathery; a few hæmorrhages may be scattered about, but the striking feature consists in the patches of white exudate large or small, irregular in shape and distribution, or arranged symmetrically about the macula: such is the picture of an albuminuric retinitis, so-called. But not infrequently we meet with cases which are not only difficult to place, but which, while resembling one or another of these two clinical types, are the result of some totally different cause.

Again, these two clinical groups are quite distinct in the significance which



they bear with respect to the prognosis of life. In the case of the arterio-sclerotic type we know quite well that the expectation of life is much greater than in the albuminuric type.

In face of all these considerations I really cannot see that the views enunciated by Dr. Batty Shaw are in any way helpful in the differentiation or prognosis of arterio-sclerotic and renal retinitis. In effect he says that arterio-sclerotic and renal retinitis cannot be differentiated. Let us assume for a moment that the arterio-sclerosis and the nephritis are but different aspects or results of some hypothetical toxæmia, as indeed they may be. In one person this toxæmia produces an arterio-sclerosis, in another a nephritis. Or again, it may be assumed that one form of toxæmia is responsible for the arterio-sclerosis and another toxin lies at the root of the nephritis. But whatever may be the originating cause, they have at any rate produced two entirely different clinical varieties (I use the word clinical advisedly, as opposed to pathological), each of which has its own prognostic significance. And this, again, is in consonance with what we know of other forms of toxæmia, such, for instance, as infections produced by the pneumococcus. At one time it may produce a pneumonia, at another time a meningitis or, again, a synovitis. So that although we have a common origin, yet we have widely differing clinical pictures, each having different prognostic significance.

I will now refer to one aspect of arterio-sclerotic retinitis which has already been touched upon, the relationship between sclerosis of the retinal vessels and the condition of the vessels supplying the brain. A few years ago I read somewhere that the cerebral vessels may be distinctly atheromatous without degeneration showing itself in the retinal vessels. This led me to make a systematic examination of a number of cases of hemiplegia due to cerebral arterial disease, with a view to confirming or disproving this statement. This investigation was, unfortunately, interrupted by the war, and I have not had the opportunity of taking it up again. Of fifteen genuine cases of apoplexy which I examined, nine cases showed marked arterio-sclerosis, the most marked being in a female aged 41, who had a systolic blood-pressure of 230 mm. Hg and a trace of albumin in a urine of low specific gravity, together with hæmorrhagic retinitis. In four cases the signs of retinal arterio-sclerosis were slight, whilst in two cases the arteries appeared to be perfectly normal. One of these cases was that of a woman aged 45 who had been hemiplegic for three years, in whom the blood-pressure was only 160 mm. Hg (D. 120). The other case was that of an old man aged 71 who had had a stroke one year previously, and whose blood-pressure was only 160 mm. Hg (D. 110). But what is more germane to this discussion was the fact that out of these fifteen cases only one patient showed hæmorrhagic retinitis, the case already alluded to. Of course one has seen many cases of hæmorrhagic retinitis which have been followed by stroke at varying periods. I have not been able to gather all my cases together, for these fifteen cases represent only workhouse patients and inmates. It would, however, be interesting to know what is the frequency with which hæmorrhagic retinitis can be found in cases of cerebral apoplexy, and their relationship to the blood-pressure.

Mr. M. S. MAYOU

exhibited a series of pathological slides showing the various changes in the retinal vessels together with the different forms of exudation into the various parts of the retina. He raised only one point in the discussion—that of

nomenclature. It was proposed to manufacture a new term: arterio-sclerotic retinitis. Pathologically, it was not a retinitis, but a degeneration, and if a new term was introduced, care should be taken that it was a correct one, e.g., vascular sclerotic degeneration of the retina.

Dr. G. NEWTON PITT (President of the Section of  
Medicine)

said it was very essential in the discussion of this question to draw attention to what had been pointed out years ago; that the vascular changes which took place in the aorta, in the small vessels, and in the arterioles were quite independent, each of the others. Though one set of vessels might be diseased it did not necessarily follow that all vessels were affected. There was a tendency to assume that, in cerebral hæmorrhage, for example, there was an association with arterio-sclerosis. But when examining cases post mortem, one found that there were a large number of cases of cerebral hæmorrhage in which the arteries, including the middle cerebral, did not show extensive disease. It was true that where there was extensive disease of arterioles there was more likely to be disease in the middle-sized arteries as well than if the arterioles were healthy; but the diseases of the two were distinct, and the presence of one did not justify a presupposition of the existence of the other. That was a very important consideration, because the same applied in regard to the kidney changes in cases of cerebral hæmorrhage. The number of cases of cerebral hæmorrhage which had also marked interstitial nephritis was very moderate, or comparatively so, though statistics varied as to the exact proportion, continental figures being lower than our own. What he wished particularly to insist upon was, that arterio-sclerosis must not be definitely correlated with cerebral hæmorrhage: though they might co-exist in the same patient, they should be regarded as separate entities.

The same applied to the retina: its vessels corresponded closely in size to those from which cerebral hæmorrhage occurred and were the only ones open to inspection which gave an indication of the condition of the walls of the arterioles in the brain. The figures Dr. Ellis gave at the first meeting on this subject showed that, taking the early cases of retinitis in which there was no evidence of inefficient kidney function and no excess of blood urea and in which the kidney functions were fairly good, the majority of the patients died of cerebral hæmorrhage. The cases having excessive blood urea and evidence of very inefficient kidneys were more likely to die of uræmia. What was required to be known was, the relation between cases in which there was death from cerebral hæmorrhage and those in which death occurred with retinal changes. At present, very few figures on the subject were available, as in the wards the retinæ were not systematically examined.

Whilst there were cases in which one felt that the retinal change indicated renal change, there surely must be many cases in which, to a physician, it was doubtful whether one was to look upon the change as vascular, or as renal. What was the experience of ophthalmologists on this point? Did they claim that the two were quite distinct? There were some cases which were definite, but there were a large number of cases with retinitis in which one would not like to say what was the state of the blood urea or the condition of the kidney, without making further examination.

The occurrence of cerebral softening and cerebral hæmorrhage in these retinal cases raised this question: Were the lesions due to rupture of a minute

vessel, or to a thrombosis? When a vessel was thrombosed there was an infarct, and in many of the cases in which a small vessel was blocked there were present the conditions for a small extravasation of blood just as much as if the vessel had actually ruptured. When a small retinal hæmorrhage occurred, he was therefore not at all sure that this indicated the rupture of a vessel. In many cases such hæmorrhages were more indicative of thrombosis.

There was no doubt that the prognosis of the conditions was distinct. Where there were retinal changes with distinct evidence of kidney inefficiency, the prognosis was infinitely more grave than when retinal hæmorrhages occurred and the kidneys were efficient. Personally, he would be much more inclined to base his prognosis on the condition of the blood urea and the kidney efficiency than simply on the appearances in the retina.

A very small proportion of cases with fatal uræmia due to interstitial nephritis showed retinal changes, and in the most extreme forms of the lesion often no retinitis developed. What was the additional factor which determined that in a small proportion there should be retinal changes, and in the majority not? There were not sufficient data available for forming a definite conclusion as to this; but merely to assume that there was a toxic condition which had produced fatal nephritis did not sufficiently explain the fact that only in a small proportion of the cases would there be this retinal change.

With regard to the blood-pressure: he did not think it followed that if there were arterio-sclerotic changes in the middle-sized arteries, the pressure in the capillaries and the arterioles was necessarily raised. In many of the cases having arterio-sclerotic vessels it was a question whether the tissues were not suffering from too low rather than from too high a pressure. And there was much evidence favouring the view that these conditions were due to defective nutrition, and that it was a cutting off the blood supply which caused exudations, as well as small hæmorrhages.

#### DR. A. FEILING

said the remarks he would make were based essentially on the study of thirty cases in the last eighteen months, and they had been observed from the point of view of the physician, not from that of minute changes in the fundus oculi. All those thirty cases were referred to him by his ophthalmic colleagues, and all sought advice in the first instance because of failure of vision, not for symptoms referred to any other system of the body. For this discussion, he tried to divide the cases into those which he would call arterio-sclerotic, and those he would designate renal. In the majority he found it fairly easy, on clinical grounds, to do so. The clinical symptoms he took for differentiation were: (1) A history of any definite attack of acute nephritis; (2) the persistent presence of large amounts of protein in the urine; (3) the presence of well-marked œdema. Cases presenting all those characteristics he classified provisionally as renal. And when the cases were followed into detail, they all corresponded to the renal group, to which Mr. Foster Moore drew attention.

Of the thirty cases, he classified only five as renal—three males, two females. The average age was 43·8 years, and all had well-marked bilateral retinitis. One had had nephritis during the war, i.e., in 1918, and was for nine months in hospital before he was considered well enough to be discharged. The second patient said he was in a London hospital under Dr. Pavy twenty years ago for acute nephritis. In the other three he was unable to get a definite history of acute renal disease, yet he did not think anyone would hesitate to class them as renal. Of the renal cases, the average systolic blood-pressure was 235 mm., and the diastolic pressure 135.

In the group he called arterio-sclerotic, there were several points of difference which were of great interest. Their average age was 63·3 years, which was in contrast with 43·8 years in the renal group, and fifteen of the twenty-five were females. Of the twenty-five, 60 per cent. had the retinitis unilaterally. The average systolic pressure was 214, the diastolic 118. In both groups there was high arterial blood-pressure, well-marked thickening of the accessible arteries, and, generally, some hypertrophy of the heart which was evident upon examination by ordinary clinical methods.

After discussing various hypotheses, illustrated from his own experience, Dr. Feiling concluded that there should be hesitation about adopting any new nomenclature in these conditions; the toxic idea was only at present based on theory, and to adopt the suggested new nomenclature implied the risk of shutting one's eyes to other causes, such as the mechanical one.

#### MR. J. HERBERT FISHER.

Referring to Dr. Batty Shaw's opening address, I was particularly interested in his advocacy of the substitution of such terms as "minimal and maximal toxic retinitis" for arterio-sclerotic and renal retinitis. In 1915 I read a paper before the Section of Ophthalmology on the retinitis of pregnancy, in which I advocated the use of the term "toxæmic retinitis of pregnancy" instead of albuminuric retinitis of pregnancy. After all, we are in the midst of a discussion, and some nomenclature has to be adopted unless our efforts are to come to an abrupt conclusion. The obstetrician has advanced reasons for believing that the pathological vomiting of pregnancy, eclampsia, acute yellow atrophy of liver and the necrotic changes of the kidney cortex that accompany the albuminuria of pregnancy are due to a toxin, and has suggested that the toxin may be produced by perverted katabolic processes in the syncytium cells shed from the chorionic villi at the placental site into the maternal circulation. In all the various organs liable to attack, the stress of the lesions is upon the blood-vessels, and hæmorrhages in consequence are a conspicuous feature; and it seems at least reasonable to infer that the fulminating lesions in the retina in these cases accompanied by exudates, hæmorrhages and œdema, are due to the same cause, and that in considering this variety of retinitis at any rate we are getting pretty close on the scent of the nature and source of the toxæmia.

At the other end of the scale it seems legitimate to take a glance at a variety of retinitis in which the natural changes of advancing years produce such alterations in the coats of the blood-vessels of the retina that exudates in the retina, based on hæmorrhage, result; I refer to retinitis circinata—a disease of the later years of life—so chronic in character that its explanation on the basis of the altered condition of the arterial tunics seems to fulfil every requirement without invoking any conception of an absorption toxæmia. Nearly twenty years ago I brought before the Ophthalmological Society<sup>1</sup> a boy, 13 years of age, who after acute rheumatism eight years earlier had developed cardiac disease, and showed multiple aneurysmal dilatations on some of his retinal arteries, clearly indicating very advanced changes in the arterial walls; in his retina he presented an appearance of glistening exudate entirely comparable to the senile cases of retinitis circinata. More recently, at a combined

<sup>1</sup> *Proceedings*, 1915, viii (Sect. Ophth.), pp. 127-148.

<sup>2</sup> *Trans. Ophth. Soc. Lond.*, 1903, xxiii, p. 73.

discussion on "Diabetic Retinitis," a striking fact was confirmed, viz.: that diabetic retinitis was rarely manifested in the most serious and fatal cases of diabetes which attacked patients in the first half of life, while it was frequent in the less serious cases of glycosuria occurring in the later half of life. It would appear that the toxæmia was most intense in the cases in which retinitis failed to manifest itself, and it was generally agreed that the milder toxæmia of the other group was capable of producing the retinal changes by reason of the sclerotic changes in the blood-vessel coats which had resulted from advancing years.

Most of the speakers in the present discussion have agreed that there is an arterio-sclerotic retinitis, as well as the more familiar retinitis, which, for the moment at any rate, we must still designate as an albuminuric or a renal retinitis, and for the most part they are agreed that it is not in many instances possible to distinguish absolutely, from the ophthalmoscopic appearances, between the one and the other. Dr. Batty Shaw inclines to the view that whether albuminuria be present or absent the cause is a toxæmia. It has been shown that in one group of cases in which the kidney functions well the prognosis for life is by no means so dismal as in the other. As an alternative to Dr. Batty Shaw's view, it has been suggested in the course of the discussion that in the unfavourable cases interstitial fibrosis of the kidney initiated by some acute nephritis is the primary disorder, and that the cardiac hypertrophy, with the resulting arterial thickenings, is a compensatory phenomenon, and assists the impaired renal tissue to function, though still indifferently; while in the cases which are arterio-sclerotic and attributable to some form of as yet unrecognized toxæmia, the cardiac hypertrophy must necessarily follow to drive the blood-stream against the increased resistance, but, that with this assistance, a reasonably sound kidney still functions well and serves an admirable purpose by eliminating the toxins from the blood. Dr. Batty Shaw appears to incline to the view that the toxin is likely to be the same but of different intensity in the two varieties of disorder, both of which are capable of producing retinal changes, and these changes to a large extent indistinguishable. Such a view seems to derive some support from the fact that though life may be much more prolonged in the cases in which there is no albuminuria, yet the causes of death, when it comes, are at any rate in many instances identical with those which produce death in a comparatively few months after retinitis develops in the presence of kidney insufficiency. If we accept the toxic explanation of both varieties, it appears reasonable to expect that in the arterio-sclerotic cases with efficient kidneys, the toxin should be found abundantly in the urine, but in low concentration in the blood; while in the albuminuric cases, where elimination of the toxin by the kidney is impeded, the toxin should be found in concentration in the blood, but sparsely in the urine. Might it be that comparative records of analysis of blood and urine in the two classes of cases by chemical pathologists will put us on the track of a toxin which at present is hypothetical?

It appears, therefore, to me that in the retinitis of pregnancy we have a manifestation which is typically toxæmic in origin; that retinitis circinata affords an example of a retinitis with hæmorrhages dependent on sclerosis of retinal vessels; that in diabetic retinitis we see the effects on the retina of specific chemical toxins capable of operating only in the presence of those changes in the walls of the blood-vessels which are common to all mankind as age increases. That in arterio-sclerotic retinitis and in so-called renal retinitis we have yet to discover a toxin, which quite probably may be common to both,

and which is in greater intensity in the blood and body tissues and likely therefore to prove destructive to life at an early date only if the kidney is impaired in its function as an organ of elimination.

Dr. C. F. HARFORD.

It seems to be clear from the introductory paper of Dr. Batty Shaw and from subsequent speakers that there is no such close relation between changes in the retina and disease in the kidneys as common tradition has sketched for us. This being so, it is our plain duty to review this subject from every point of view and in the meantime we should refrain from giving the grave prognosis which has previously been suggested, as we may by this very act be accelerating the fatal issue which we desire to avoid.

It would have been most instructive if Mr. Foster Moore could have given us a series of pictures of the fundus in cases which he describes as arterio-sclerosis and those which he refers to as renal retinitis. These would have afforded us an opportunity of considering anew whether the appearances in the two classes of cases could be regarded as distinctive apart from the clinical picture presented by the physician. He (Mr. Foster Moore) has told us that the signs which he describes as due to arterio-sclerosis cannot be looked upon as pathognomonic. Should we not be right to conclude from this that the appearances in question are common to many forms of disease which give rise to changes in the peripheral part of the vascular system, such as in the kidney and in the retina, each of which possesses a highly specialized arrangement of its terminal vessels? It will be noted that I have not referred here to cardio-vascular changes, for the questions of high blood-pressure and the like involve quite a different aspect of the case. Dr. Ellis, with his interesting series of cases, has afforded us valuable material for thought, as he has given us the benefit of the most recent methods of testing renal efficiency.

I will quote here a passage from the paper by Professor Hugh MacLean, delivered at the annual meeting of the British Medical Association, published in the *British Medical Journal* for December 2, 1922, relating to cardio-vascular changes and its effect on prognosis:

"There are many subjects who show but little evidence of marked cardio-vascular changes, but in whom the renal system is hopelessly inefficient. Conversely, it is not uncommon to find patients with very marked cardio-vascular changes in whom but little evidence of renal disease can be ascertained. These points must always be taken into consideration in estimating prognosis, for, in a general way, apart from such accidents as cerebral hæmorrhage, the outlook in a patient with high blood-pressure is not so bad if the kidneys are efficient. Indeed, such patients may enjoy comparatively good health for many years, even with a blood-pressure as high as 200 mm. mercury or even more. This observation explains the curious cases one occasionally finds quoted in the literature, in which a history of high blood-pressure of 250 or over, frequently associated with retinitis and other eye changes, was not incompatible with the enjoyment of fairly good health for several years."

The conclusion to be deduced from these researches may be best expressed in the words of my old teacher, Sir Michael Foster, when he had come to an end of his lecture on some fascinating physiological problem, told with convincing force: "The matter is not yet ripe for any dogmatic statement." In spite of this we still continue to use that most dangerous weapon of prophecy, which we designate prognosis, in order to foretell the year if not the



date of a man's death. Some of us have been in touch with primitive races among whom the emotions have more than ordinary influence, and we know that the mere expectation of death in many cases is sufficient to produce this result. This condition is not unknown in human life nearer home and it should prescribe to us supreme caution in the statements which we make in public or in the secrecy of our consulting rooms. This brings me to the chief personal contribution which I can make to the discussion on this subject, mainly from my recent studies in the psychological aspects of health and disease. A year ago I wrote a paper for the International Ophthalmological Congress at Washington, which was presented there in April last, on "Psychopathology in its Relation to Ophthalmic Practice," and in this I drew attention to the close connexion between diseases of the eye and those which were looked upon chiefly as psychical disorders. Since then I have been developing the hints which I then put forward in many directions.

I propose to make two suggestions bearing upon the present discussion in the hope that they may serve as a fruitful germ of thought to others. (1) The element of *cardio-vascular disturbance* is one of the essential factors in the fatal termination in cases such as those we are considering. Without taking special account of the precise effect upon the pulse or the blood-pressure of what we may call an emotional trauma, we are all of us aware of the disastrous results of a condition of panic or passion. The blanching of the skin of the face in the one case and the reddening in the other indicate the effect on the peripheral circulation of these emotions, which may be associated with violent physical manifestations. Thus in the case of panic there may be a state akin to temporary paralysis, or in passion violent muscular tremors. These may be said to be attributable to an element of fear, which it is the duty of the psychologist to investigate. It is contended that phobias of varying amount are an essential element in advanced cases of renal disease or other serious illnesses with a reputation for incurability, especially when the severity of the case appears to be indicated by ocular complications; and this needs to be borne in mind.

(2) With regard to the changes in the peripheral circulation, let me assume that Dr. Batty Shaw's hypothesis is correct, and that we are dealing with a toxic process affecting both the kidneys and the retina. The problem which is always before us is the explanation of the selective action of various toxins producing changes in the various vital organs. A great deal of evidence is available to prove the profound influence of suggestion or auto-suggestion upon local inflammation and vascular disturbances, especially of the skin. It is contended that this is due to the effect upon the peripheral circulation of psychical processes acting through the medium of the nervous and circulatory mechanism, and that suggestions of an unfavourable kind tend to produce morbid vascular changes. It is impossible in a contribution to a discussion to do more than suggest the lines upon which inquiry might be conducted, and it is earnestly hoped that the teaching of modern psychology may be considered in its relation to these obscure problems of pathology.

Dr. BATTY SHAW (in reply) (read by Dr. IZOD BENNETT).

This discussion has been fruitful in at least one direction, viz., that it has given an opportunity for a statement of claim to those who believe that there is something behind arterio-sclerosis which is not only responsible for the occurrence of arterio-sclerosis, but which is independently responsible for other



changes such as those met with in the retina when the blood-vessels of that tissue *are altered* and for similar changes in the retina when such vascular change is *slight or quite absent*; and we must hope that impetus may be given to those who would look for some agent responsible for both, in the blood circulating within the vessels.

Important as the result of discussion is, it is but a part of a much larger question, for all that has been said against the view that arterial disease is responsible for so-called arterio-sclerotic retinitis may be equally urged against the view that the contracted kidney is responsible for what is known as uræmia. It will have struck other members of this audience that with the exception of five cases out of the twenty-seven tabulated by Mr. Foster Moore, in which no blood-pressure readings were made, there was only one patient in whom the blood-pressure was not raised above 150 mm. of mercury, i.e., blood-pressure was raised in 97 per cent. of the cases. If we believe that the rise of blood-pressure may result from the occlusion of the lumen of arteries by endarteritis which has gradually developed and become universal, the occurrence of hyperpiesis is readily explained, but as I have pointed out, this obliterative disease of the arteries must be capable of running an unexplainably fugitive and recurrent course, or we should not see the rapid falls of blood-pressure or the equally rapid rises of blood-pressure I have figured. If we believe, as we must be asked to do, that hyperpiesis can result from obliterative disease of only a part of the arterial tree—for all of Mr. Foster Moore's cases of arterio-sclerotic retinitis were free from the suspicion that the renal arteries at any rate were so diseased as to produce renal disease—we must re-write the physiology of the vasomotor system. At present we know that if the arterial system of such a large tract as one leg is blocked by ligature at amputation, the blood-pressure of the rest of the arterial tree is not raised; adjustment is made in the vasomotor system and the blood-pressure of the rest of the body remains normal, as observation has shown. It seems to me that the large proportion of Mr. Foster Moore's cases show hyperpiesis because there is some pressor body, or bodies, circulating in the blood-stream.

Dr. Hawthorne objects to the use of qualifications such as "arterio-sclerotic," "renal," or "toxic," on the ground that the problems underlying this nomenclature have not yet been solved; but I think it is apparent to all of us that there is little advantage in speaking of retinitis occurring in arterio-sclerosis and retinitis occurring in renal disease, or in toxæmia, for we know that authorities would still believe that there were retinal changes met with in arterio-sclerosis entirely dependent upon the arterio-sclerosis—a view so ably presented by Mr. Foster Moore, and if we speak of retinitis present in renal disease we should perpetuate the belief that even in these cases the renal disease actually causes the mischief. If Dr. Hawthorne hesitates to accept my view that a pre-renal toxæmia is responsible for both the arterial change and for the retinal change, directly in both cases, on the ground that no one has yet demonstrated the presence of such agencies, I would suggest instead of the term "toxic retinitis," "hyperpiesic retinitis," for hyperpiesis was present in nearly all of Mr. Foster Moore's cases of so-called arterio-sclerotic retinitis; and I can state that in eleven cases which formed part of my recent study, retinitis was present and they were all cases of hyperpiesis. I have given reasons elsewhere why I think such great confusion has arisen in the symptomatology of renal disease, and have tried to show how all such difficulties could be resolved if, instead of expecting to find regularly a well-defined picture of kidney disease behind each well-defined clinical picture,

we gave up the position and rather looked to the blood as a source, not only of the clinical manifestations, but of the kidney change. This point of view is brought out by Dr. Goldschmidt in his clinical report (p. 23) of a form of disease which though not identical with that under discussion is closely allied to it.

Mr. R. FOSTER MOORE (in reply).

It is clearly impossible, in the short time remaining, to touch upon more than a few of the points which have been raised by the various speakers in this debate.

I do not hesitate to agree with Dr. Batty Shaw that retinitis may be caused by toxæmia, as in the retinitis of pregnancy instanced by Mr. Fisher, and I would add parenchymatous nephritis in general; but, on the other hand, it cannot be denied that retinitis may occur from causes which are local in the retina, as for example, the star figure which may accompany papillœdema, so that it is at least not unreasonable to suppose that retinitis may be caused by the local vascular disease.

In renal retinitis there are, I believe, two factors at work, the one a toxic factor, which manifests itself by the "cotton-wool" patches, which, histologically, are found to be due to fibrinous exudate which may infiltrate all the retinal layers; and the other a vascular factor, which manifests itself by the presence of exudate having the ophthalmoscopic characters we have described in addition to evident changes in the vessels, and which is due to small areas of hyaline degeneration in the external molecular layer of the retina. The former is seen in parenchymatous nephritis, the latter in arterio-sclerotic retinitis, and I believe both these factors may be concerned in varying degree in renal retinitis.

I believe, then, that the changes in the retina in arterio-sclerosis to which the term "retinitis" may be applied are immediately due to the impaired circulation in the retina consequent upon the local disease of the vessels, but I should by no means be willing to dispute that this disease is in its turn dependent upon a toxæmia, whether of endogenous or exogenous origin. I have referred to this point in a previous paper.<sup>1</sup>

With regard to the immediate lesion which is responsible for hæmorrhages in the retina, I do not suppose they are due to rupture of large vessels, but imagine they are due to diapedesis from the capillaries, owing to the impaired nutrition of the endothelium resulting from defective circulation, and perhaps to changes in the blood. I believe that in these cases the blood-pressure in vessels of the size of the central retinal artery is less than the normal, even though the pressure in the brachial artery is perhaps 250 mm. of mercury; I suspect, too, that the retinal hæmorrhages which are so frequent in the blood diseases come about in the same way, through impaired nutrition of the capillary walls.

I was interested in Mr. Bardsley's important observation that the "copper-wire" appearance of arteries may disappear; such an observation is outside my own experience.

With regard to Dr. Hawthorne's remarks, we know what a terminological purist he is, but he is less of a purist when he tells us that the differentiation between renal and arterio-sclerotic retinitis is no more than guess work!

<sup>1</sup> *Quart. Journ. Med.*, 1917, x, p. 41.

I was very glad to find Dr. Gaskell so emphatic in his belief in a primary disease of the vessels as quite distinct from renal disease. I do not hesitate to agree with Mr. Leighton Davies that arterio-sclerotic retinitis cannot in all cases be sharply differentiated from renal retinitis. I thought I had protected myself against Mr. Mayou's criticism by pointing out that I was using the term "retinitis" in the sense that it is used in renal and diabetic retinitis; it is not a good term, but it is hallowed by long usage, and would, I fear, be difficult to dislodge; I wish Mr. Mayou would supply us with a better term—it is badly needed.

With regard to Dr. Newton Pitt's remarks, I do not think the hæmorrhages are due to the lodgment of emboli; they are different in type from the hæmorrhages which are so frequent in the retina in infective endocarditis, and which I presume are indeed due to emboli.

I was glad to hear that Dr. Feiling found, in agreement with my own figures, that 60 per cent. of his cases of retinitis in arterio-sclerosis were unilateral; this alone is surely a fact which weighs heavily against the view that toxæmia is the direct cause of the retinitis; he mentions that fifteen of his twenty-five cases were in females; this preponderance of females has been commented upon by Nettleship, Gunn and Adams and the fact of its occurrence is borne out by my own cases.

Retinal detachments are not rare in severe renal retinitis if they are specifically looked for up to the time of death; I refer to extensive bilateral detachments, and not to a collection of exudate under the retina which is of still more frequent occurrence in histological specimens, but is not to be made out with certainty by the ophthalmoscope. These conspicuous detachments are often overlooked from the fact that the physician, having ascertained the existence of retinitis, does not always continue with periodic examinations up to the time of death; I have seen two of these cases within the last six months, and was able to collect thirteen of them in two years at St. Bartholomew's Hospital.

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## Section of Neurology.

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### Observations on Myopathy.

By EDWIN BRAMWELL, M.D.

I PROPOSE to refer to some experiences and problems in relation to myopathy. While some of my observations may appear to be suggestive rather than conclusive, they will, I hope, for this reason, stimulate discussion.

#### SYMMETRICAL ATROPHIC PARESIS OF THE QUADRICEPS MUSCLES OF PROBABLE MYOPATHIC ORIGIN.

The distribution of the affected muscles in the myopathies shows considerable variation, and a number of well known types are described in accordance with the incidence of the muscular involvement. Two cases characterized by progressive bilateral weakness and symmetrical wasting of the quadriceps muscles, which are, I believe, examples of myopathy, are of interest on account of the unusual distribution and limitation of the atrophy.

*Case I: Bilateral Progressive Weakness and Wasting of the Quadriceps Muscles.*—A married woman, aged 53, complaining of weakness in the legs, was seen on October 4, 1921.

*History:* The patient, for a year previously, had noticed difficulty in rising from a chair or ascending stairs. This disability had apparently developed gradually, had been unaccompanied by pain or other subjective sensory symptom, and had been progressive. The thighs had become appreciably thinner; otherwise, the patient stated, she felt in perfect health. There was no history of any obvious ætiological factor or of any similar condition in the family.

*State:* Symmetrical wasting of the thighs, especially of the vasti interni, was noted on examination. The movement of extension at the knee-joints was very easily overcome, and when the patient attempted to bend the knees she would have fallen had she not been supported. No weakness of any movement, apart from extension at the knee, was detected. The electrical response in the quadriceps muscles showed a varying degree of quantitative reduction but no qualitative change. There were no fibrillary tremors. A feeble knee-jerk was obtained on reinforcement, but no contraction was observed in the vasti interni muscles. The ankle-jerks were brisk and equal, there was no tendency to clonus, and the plantar reflexes were definitely flexion. No subjective sensory symptoms had been complained of, and no objective sensory disturbance was found. There was no sphincter trouble and no vertebral deformity. Radiograms of the spine showed no defect in the bony arches. No abnormality of the nervous system or of the thoracic or abdominal viscera was detected. The urine contained neither albumin nor sugar, and the Wassermann reaction, both in the blood and cerebro-spinal fluid, was negative.

The leading features of another case presenting a very similar clinical picture are as follows:—

*Case II: Bilateral Progressive Weakness of the Quadriceps Muscles; Congenital Absence of the Brachio-radialis and Biceps Femoris.*—A ploughman, aged 59, who complained of weakness of the legs, was seen on November 3, 1921.

**History:** The patient stated that some six or seven years previously he had first noticed a tendency for the knees to give way if, when ploughing in wet weather towards the end of the day's work, his boots became clogged with clay. Up to this time he said he had been "very smart on his feet," and could run and jump as well as any man of his age. About the same time he found that he was unable to walk upstairs carrying a heavy load on his back. A year or two later he found that when he bent his knees beyond a certain point he would collapse in a sitting position. At a later date he was only able to go upstairs one step at a time, and recently when ascending stairs he had been obliged to hold on to the banister. No history of any similar condition in the family was obtained.

**State:** The patient looked rather older than his years. His general muscular development was good. There was pronounced symmetrical wasting of the thighs, particularly in the lower two-thirds; the tensor fasciæ femoris muscles, on the contrary, were unusually well developed. Extension at the knee was very easily overcome. In rising from the ground and assuming the erect position, the patient was obliged to support himself by placing his hands upon his knees. The biceps femoris muscle could not definitely be demonstrated, and the brachio-radialis (supinator longus) muscle was absent on both sides. Apart from the weakness of extension at the knee, all the other movements of the body were powerfully carried out. The electrical examination showed a quantitative reduction in the wasted portion of the quadriceps muscles. No abnormality of the nervous or muscular systems was otherwise detected; no fibrillary tremors were observed; the knee-jerks were absent, while the other reflexes presented no abnormality. Neither pain nor other subjective sensory disturbance had been complained of. There was no sensory loss, no sphincter trouble, and no evidence of vertebral defect or disease. Movements at the hip-joints were quite free. The Wassermann reaction was negative. The arteries were slightly thickened and tortuous, but the heart was not enlarged, and the blood-pressure not unduly high. There was no polyuria, and the urine was free from albumin and sugar. The conformation of the abdomen suggested a degree of visceral ptosis, and a bismuth meal showed some delay in the large bowel. Sir Harold Stiles, whom I asked to examine the patient in relation to the condition of the muscles, kindly sent me a detailed report, from which I abstract the following remarks: "There is visible wasting, more especially in the lower quadriceps region of both thighs; the whole of these muscles are either partly atrophied or imperfectly developed. If one assists the patient to adopt the squatting position, he is quite unable to raise himself into the erect posture without great assistance, owing no doubt to the imperfect action of the quadriceps. I was unable to discover any functional impairment in any of the other muscles of the lower extremities (with the exception of the biceps femoris). I formed the opinion that the tensor fasciæ femoris had become hypertrophied to compensate for the weakness of the quadriceps, and I was inclined to think that the anterior fibres of the gluteus medius and minimus muscles had also participated in this hypertrophy. All the muscles of the upper extremities appear to be normal, with the exception of the brachio-radialis (supinator longus) muscles, which are either congenitally absent or so imperfectly developed as to be quite undemonstrable."

The two cases I have just described resembled each other closely. In both there was a symmetrical, atrophic and apparently progressive palsy, limited to the quadriceps muscles, with a quantitative reduction in the electrical excitability, and great diminution or loss of the knee-jerks. Again, no fibrillary tremors were observed, nor was there any subjective or objective sensory disturbance. Further, it is interesting to note that both patients were in the sixth decade of life. These cases, I submit, are to be regarded as examples of myopathy in which the process is limited to the quadriceps muscles. Although it is affirmed that the anterior thigh muscles may be the first to be affected in a case of myopathy, I have not hitherto met with a case in which these muscles were alone implicated.

The second case differs from the first, since in the former the supinator

longus and biceps femoris muscles were absent, while the tensor fasciæ femoris and the anterior fibres of the gluteus medius and minimus were abnormally well developed. The hypertrophy of the tensor fasciæ femoris and anterior fibres of the gluteus medius and minimus was, from its distribution, obviously compensatory.<sup>1</sup> There can, I take it, be little doubt that the absence of the supinator longus and biceps femoris is to be ascribed to a congenital defect. Indeed the question arises whether the disability might not be explained by a congenital muscular defect, the apparent progressive weakness being accounted for by the supervention of the general deterioration of the muscular system, which occurs with advancing years. While this possibility cannot perhaps be excluded with certainty, my strong impression is that there has been progressive localized weakness in the quadriceps group, for the patient, who was a muscular man, stated emphatically not only that up to the time he first noticed his disability he could run and jump as well as any man of his age, but that since that time his weakness had been steadily, if slowly, increasing.

Congenital anomalies of muscle, I may remind you, have been noted by several observers in the myopathies. Thus cases of the kind, in which muscles were congenitally absent, have been reported by Erb, Abromeit, Ziehen, and others. The late Dr. Batten remarked that a child might be born with a myopathic facies and yet for many years there might be no further developments. Apart from congenital absence of muscles, other muscular anomalies in development have also been described in cases of myopathy. Oppenheim, for instance, refers to a case in which he found a muscle with supernumerary digitations. Again, in two cases of the scapulo-humeral type, I observed a bilateral anomaly of the biceps muscle, with which you are very possibly familiar, and which I take it must be of congenital origin. When these patients voluntarily flexed the arm at the elbow, the biceps stood out as a round mass opposite the insertion of the deltoid muscle, while little if any contractile tissue was demonstrable below this point. The facts to which I have referred would appear to indicate that there is a tendency to congenital muscle defects in the myopathies. Other congenital stigmata, on the contrary, do not appear to belong to the picture of these disorders, although in this respect myotonia atrophica, which is commonly classed with the myopathies, is quite exceptional. A congenital muscular anomaly in a case of doubtful myopathy is, I submit, comparable in its significance to that of a spina bifida occulta in a case of central gliosis.

#### MUSCULAR DYSTROPHY AND TRAUMA.

Does trauma ever play a part in determining or aggravating a myopathy? This is a question which may come to be a matter of practical importance, as in two cases to which I shall now refer.

*Case III: Myopathy with Facio-scapular Distribution attributed to an Alleged Trauma.*—A soldier, aged 24, complaining of weakness in the arms, was seen upon September 27, 1916.

*History:* The patient had been enlisted on August 31, 1914, as an A1 man. He stated that before enlisting he had been in the best of health and that up to that time he had been doing a full day's work as a ship's plater. No comment was made, he said, as to his appearance either in August, 1914, or in October, 1915, when

<sup>1</sup> Hypertrophy of the tensor fasciæ femoris muscles was a striking feature in a case of myopathy of the juvenile type which has since come under my notice. The anterior thigh muscles were otherwise much wasted.

he was examined, stripped, by the medical officer before entering the firing line. In February, 1916, he had been buried, he told us, in a trench and had lain on his face covered by earth and sandbags for about an hour before he was extricated. Although he did not go sick at the time, the patient stated that for some months after this accident he was troubled with a good deal of pain about the upper part of the back and shoulders. Further, he told us that in May, 1916, some three months after he had been buried, he had first noticed some slight weakness of the arms, and that in June, when he reported sick with trench feet, the medical officer to whom he mentioned these pains drew attention to the peculiar appearance of his shoulders. When, after his discharge from hospital in September, 1916, he was returned to his depot, he stated that because of the weakness in the arms he could no longer handle a rifle. He was consequently sent to hospital for examination.

State: The patient was an exceptionally muscular man with very sloping shoulders. It was noted that the sternal portion of both pectorals was absent, and the clavicular portion, especially on the right side, very poorly developed; the upper third of both trapezii was much wasted, and the lower two-thirds undemonstrable; the latissimus dorsi on both sides was very weak and atrophied. The scapulae projected to some extent when the arms were held horizontally forwards, but the serrati were acting powerfully. The spinati, rhomboids, sternomastoids, teres major and erector spinae were all very well developed. There was no weakness or wasting of upper arms, forearms, lower limbs or trunk. The tendon reflexes were present and equal; the plantar reflexes were not elicited: there was no sensory loss. A symmetrical facial paresis of the myopathic type was present (the patient had not previously noticed anything amiss with his face). The patient could not forcibly close his eyes, he could not pout his lips, and he stated that he had never been able to whistle. Otherwise the cranial nerves showed no abnormality. Three brothers and two sisters older than himself, and two younger sisters, constituted the family. So far as the patient was aware, none of his seven brothers and sisters nor any other relative had suffered from any similar condition. When this man was examined again upon May 8, 1922, I failed to satisfy myself that the condition had definitely progressed. The patient informed me that he had been engaged in light work since his discharge from the Army.

This was obviously a case of myopathy affecting the face and certain muscles of the shoulder-girdle. Had the condition been determined or aggravated by trauma? Although the patient stated that he had noticed no weakness before enlisting and he had been accepted as an A1 man in 1914, it does not follow that the myopathy had developed since his enlistment for it is to be remembered that a myopathy may exhibit obvious evidence of its presence long before the subject complains of any consequent disability. Indeed in this case the wasting of the muscles was so extreme that it was almost impossible to believe that this could have developed in the course of a year or two, particularly since the impairment of movement complained of was comparatively slight. Hence it appeared almost certain that the myopathy had been in existence for a long period of time. The question of aggravation still, however, remained to be considered. Since no mention of the alleged accident was to be found in the patient's case sheets the veracity of his statements as to the trauma, to which he attributed his condition, may be called in question. The patient's account of the incident and his whole bearing were however such that I felt satisfied that his story was to be accepted as correct. Admitting this to be the case, several possibilities arose for consideration. Had there actually been an aggravation of the condition as the result of the accident, with a result that for the first time the patient had become conscious of a manifest interference with movement? Might it be that the pains from which the patient stated that he had suffered for some time after the accident had resulted in some impairment of movement which the patient attributed in part to muscular weakness? or was it possible that when

the medical officer drew attention to the nature of his trouble, the patient either recognized his weakness for the first time or decided to utilize the information thus obtained in order to obtain discharge. True, the patient stated that he had complained of weakness to the medical officer before the latter examined him, but was this statement correct? The circumstance that when I examined this man six years later I was unable to satisfy myself that there had been any distinct tendency for the condition to progress would seem to militate against the view that the trauma, admitting this to have occurred, had aggravated the pathological process.

The record of another case in which a progressive muscular atrophy, which I believe to be a dystrophy, was attributed to trauma, is as follows:—

*Case IV: (!) Muscular Dystrophy determined by Trauma.*—Miner, aged 53, was seen on August 7, 1913.

*History:* The patient stated that upon May 6, 1911, he had "racked" (strained) himself when shovelling coal; that he had been troubled from that time onwards with pain between the shoulder-blades; that in consequence he had been unable to work after the date mentioned; and that in the autumn of 1911, six months after the accident, he had first noticed some weakness of the shoulders which had gradually progressed, and had been followed at a later date by some weakness of the legs. The patient appeared to be an honest, straightforward man who was in no way attempting to mislead, and I was satisfied that his statements were to be relied upon so far as his observation was concerned.

*State:* Upon examination I found complete paralysis of the right and pronounced weakness of the left serratus magnus with some weakness and wasting of both sides of the trapezius (I have no note as to whether the whole of the latter muscles was involved). There was, too, distinct weakness and wasting of the latissimus dorsi and pectorals especially on the right side, but no paresis or wasting of the deltoids, upper arms, forearms, hand muscles or face. The weakness of the shoulder muscles at the time the patient was examined was undoubtedly such as to unfit him for work. The thighs were somewhat small as compared with the calves (although I have no note as to weakness of any particular groups of muscles in the lower extremities, I would almost certainly have recorded this had it been observed). When the patient lay on his back and was asked to get up, he turned over on his face and "climbed up his thighs." There were no fibrillary tremors. The muscles were not examined electrically. The knee-jerks were present and not exaggerated, and the plantar reflexes were flexion. No pain or other subjective sensory symptom was complained of and there was no sensory loss. There was a slight degree of kyphosis which was no doubt occupational. I have not been able to trace this patient and know nothing of the subsequent history of his case.

The question arose as to whether the condition of the patient was attributable to the injury upon May 6, 1911. In support of the contention of a relationship of cause and effect were the facts that the man was doing a full day's work up to the date of the accident; that he had not worked since; that according to the patient's statement he first noticed weakness of the muscles of the shoulders six months after the accident; that some weakness and wasting of the shoulder muscles had been observed when he was medically examined in February, 1912, nine months after the accident; and that there was good reason for believing that the weakness had been slowly progressive since that time. Although I was of opinion that the muscular atrophy in this case was of the nature of a dystrophy, I knew at the time of no similar cases in the literature in which trauma had been suggested as a determining or aggravating factor, as the facts in this case would seem to suggest. This case however presents a somewhat similar symptomatology to the cases described by Claude and others, in which the condition was attributed to war injuries.



A number of cases of myopathy are reported in which a trauma is affirmed to have played an ætiological rôle. Although I have made no attempt to search the literature exhaustively, I may briefly refer to some of these. "A Case of Juvenile Muscular Dystrophy in a Man after Trauma" which was demonstrated by August Hoffmann at a meeting of the Rhenish-Westphalian Medical Society held at Düsseldorf in 1904,<sup>1</sup> is of interest in this connexion. The patient, a labourer, aged 41, had been severely scalded over the arms and back. From this time onwards he stated that he had noticed progressive weakness in the upper arms and back, with the exception of the deltoids, which were hypertrophic and the supra- and infraspinati which were preserved. The pectorals were almost absent and the nates and thighs were distinctly atrophied. Although the history suggested that the dystrophy had been determined by trauma in this case, Hoffmann in a short communication made a few weeks later stated that he had since discovered that the patient had been exempted from military service twenty-two years previously on account of paralysis of the scapular muscles. It would appear that the dystrophy had remained stationary for twenty years, and during that time the man had done hard work. After the accident the condition had become rapidly worse. Consequently the dystrophy had not been determined although probably aggravated by the trauma. Hoesslin<sup>2</sup> in the same year reported the case of a youth aged 21 in whom two years after a trauma to the right shoulder a dystrophy developed, affecting the right, and later, the musculature of the left scapular region. An exhaustive inquiry showed, however, that some slight muscular weakness had existed before the trauma, and that the muscular weakness had merely progressed more rapidly after the injury. In this case, as in that reported by August Hoffmann, there was no heredity. Again, Erb in his work on "Juvenile Dystrophy" describes the case of a man in whose family there was no history of myopathy, who, at the age of 34, was rendered unconscious as the result of an accident in a stone quarry, and who a year and a half later developed a typical muscular dystrophy. Another case reported by Hitzig, and quoted by Erb, was that of a boy aged 13, who, following a blow in the region of the right temple, developed progressive weakness of the right, and three years later of the left, arm, with a typical picture of dystrophy. In this case there was no hereditary history. Two further cases are referred to by Erb. In one of these, reported by Boye, the symptoms of dystrophy were stated to have commenced in the right arm three months after an injury in the region of the right supraclavicular fossa. The other case, recorded by Israel, was that of a healthy labourer, aged 37, who sustained a severe contusion of the left hip which necessitated confinement to bed for three weeks. The dystrophy which was present at the time of examination was said to have commenced at the time of the injury and to have been accentuated by a second trauma. No hereditary history was forthcoming. Spiller, in introducing the discussion on the myopathies at the London meeting of the International Medical Congress of 1913,<sup>3</sup> reports an interesting case:—

It was that of a man, aged 19, who fell from a scaffold, a distance of 60 ft., and possibly struck his shoulders. He was able to resume his work the same day but the following day his arms were stiff and painful and remained so for two days. From that time he could not lift anything and could do only light work. He noticed about four months later that the muscles of both shoulders were wasting and that he had difficulty in using them. The muscles wasted, and when seen a year after the accident

<sup>1</sup> *Münch. med. Wochenschr.*, 1904, li, p. 1027.

<sup>2</sup> *Ibid.*, 1904, li, p. 1156.

<sup>3</sup> *Trans. Seventeenth Internat. Congr. Med.*, sect. xi, Neuropathology, pt. i, p. 129.

he could raise the upper limbs only to the level of the shoulders. The deltoid, biceps, triceps and pectoral muscles were much atrophied. No fibrillary tremors were observed and no sensory disturbance for touch or pain. The patellar reflexes were present but not increased. There was no family history. A positive Wassermann reaction was obtained. "The atrophy conformed to Erb's juvenile type and apparently had been started by the trauma, which is an interesting fact" (Spiller).

The case reports to which I have referred do not appear to afford definite proof that a true myopathy is ever determined by trauma. Indeed, a perusal of the records which I have been able to find in a cursory study of the literature indicates the caution which must be exercised before a conclusion as to a relationship of cause and effect can be legitimately arrived at. In order to establish such a relationship there must be definite proof: first, that the case is one of muscular dystrophy; secondly, that there has been a trauma of some severity; thirdly, that there was no evidence of muscular weakness preceding the accident; and, fourthly, that the symptoms of myopathy had developed within a comparatively short time after the trauma. The possibility of coincidence must always be borne in mind. In relation to this problem I would again emphasize the fact that a myopathy may exist long before the muscular defect becomes so pronounced as to give rise to occupational inconvenience. Thus Hoffmann's patient, who had been exempted from military service on account of wasting of the scapular muscles, was able to continue the arduous work of a labourer for twenty years after this date. The case of a man, aged 61, the subject of a pronounced facio-scapulo-humeral myopathy, at present under my care in hospital, illustrates the same point. This patient, who is still able to follow his occupation, that of a plumber, informs me that although his unusual build and sloping shoulders have attracted attention since boyhood, it was only some seven years ago, since which time he has found increasing difficulty in working with his arms above his head, that he has been conscious of any muscular disability.

While the evidence as to an aetiological relationship of trauma to true myopathy is, I think, to be regarded as inconclusive, it is necessary in passing that I should remind you of the group of cases to which attention has been drawn by Claude, Vigouroux and Lhermitte.<sup>1</sup> These cases, in which the pathological condition followed a bullet or shell wound, were characterized notably by a unilateral or bilateral wasting of the trapezius and serratus magnus muscles and, it may be, other muscles of the shoulder girdle. On account of the distribution of the wasting, the purely quantitative changes in the electrical responses, the normal condition of the reflexes and sensation, and the fact that the position of the wound was not such as to interfere with the nerve supply to these muscles, the authors class these cases under the term muscular dystrophy of the myopathic type. They regard the condition as due to a development under the influence of trauma of an "impairment of nutrition of certain muscles giving rise to atrophy and secondarily to paresis." Huet and Français have described somewhat similar cases following war injuries. Possibly the last of my series of cases (Case IV), Spiller's case, and some of the other cases referred to are of a similar nature. These cases, however, would appear to depend upon a pathological process quite distinct from the true myopathies. Madame Athanassio-Benisty<sup>2</sup> in discussing Claude's cases remarks:—

"We have seen three cases of unilateral paralysis of the trapezius and serratus magnus, the first following an attack of rheumatism of the shoulder (according to the

<sup>1</sup> *Presse médicale*, 1915, xxiii, p. 393.

<sup>2</sup> "Treatment and Repair of Nerve Lesions" (Translation). Edited by E. Farquhar Buzzard, 1918, p. 156.

diagnosis on the case sheet), the second from carrying a heavy burden fixed by a strap to the shoulder, and the third which had occurred after a wound in the scapular region. The symptoms were identical with those described by Claude, Vigouroux and Lhermitte."

The association of isolated unilateral paralysis of the serratus magnus and the lower portion of the trapezius muscles is well known. In 1903, J. W. Struthers and I recorded such a case in which the symptoms were first noticed six days after an operation for appendicitis and in which we could obtain no certain evidence of trauma. At that time we were able to find references to seventeen cases of paralysis limited to these muscles, and I have seen several examples since.

#### MAY RECOVERY TAKE PLACE IN CASES OF MYOPATHY?

This problem was discussed by the late Dr. F. E. Batten in the *Quarterly Journal of Medicine* for April, 1910. After reviewing the literature of reported recoveries, Batten selected three cases which, in his opinion, might be regarded as instances of myopathy in which recovery had occurred. Thus of a case reported by Marina he says: "This case must, I think, be accepted as a case of myopathy in which recovery had taken place." Regarding Jendrassik's case he remarks: "In this case, again, the evidence for the diagnosis of myopathy might be criticized, yet it is difficult to see in what other group the case could be placed." In Erb's case he thinks "the diagnosis can hardly be called in question." (Spiller in his introductory paper on the relation of the myopathies at the London meeting of the International Congress refers to a case of myopathy in "an adult in which so great improvement occurred that it amounted to recovery.")

The case which I am about to report is of particular interest and importance since the patient was examined by Dr. Batten.

*Case V: Recovery in a Case diagnosed as Myopathy (! Toxic Neuritis).—W. C., aged 5, an only son, one of a family of four, one sister being older and two sisters younger than the patient, was seen on September 26, 1919.*

*History:* As a baby the patient had appeared to be quite healthy, although his mother stated that when placed on his back he had unusual difficulty in turning over. I have no note as to when the child first began to walk, but according to his mother he showed a tendency to waddle when walking about Christmas, 1917, i.e., two years before I saw him. The onset would seem to have been gradual. In May, 1918, the boy was seen by Dr. Batten. I have a letter written to the patient's mother upon May 20, 1918, in which Batten expressed himself in the following terms: "Although I must admit that there is much in favour of the diagnosis of pseudo-hypertrophic paralysis already given you, yet there are several unusual features which make me hesitate to give that diagnosis unreservedly, and I should be prepared to consider the possibility of a toxic neuritis, from which recovery is possible after a period of about eighteen months or two years. I very much want to find out about the family history. If it can be shown that the members of the family suffered from a form of paralysis which corresponds in course and type with that now occurring in W., then I fear it would not be possible to accept any other diagnosis than that of myopathy."

The patient's mother informed me that Dr. Batten, after examining the boy again, had written her that he was satisfied that the diagnosis of myopathy was correct, and that only three cases were known in which recovery had taken place. Unfortunately I have not the letter in which Batten expressed this opinion. The patient's medical attendant wrote me, on March 24, 1922, as follows: "I am sorry not to be able to send Batten's last letter, which would have helped you more than these notes; there was a later one which the boy's mother has mislaid, but which I saw, in which he stated that he was inclined to change his opinion, and regarded the condition as a

myopathy. I may say that there was no family history of importance. The first time that Sir Harold Stiles saw the boy he was a typical pseudo-hypertrophic in gait and manner of getting up from the floor, and laxity of the shoulder girdle when lifted with one's hands in the armpits. Professor Gulland also saw him on the same day and diagnosed myopathy."

The child's mother stated that on August 2, 1919, she took him to a herbalist, from whom he had been receiving treatment since. She further told me that the boy soon began to improve under this treatment, whereas the massage and electricity he had been having previously had produced no effect. On the other hand, the patient's doctor writes me, on March 24, 1922: "The old herbalist woman gets the credit for a cure, but he had begun to improve before that."

State: My notes at the time I examined the patient (September 26, 1919), were as follows: "The boy is very active and alert mentally. When he walks there is a slight lordosis and a distinct tendency to waddle; he cannot stand securely on his toes, nor can he raise the toes from the ground and walk on the heels; if he bends his knees to any extent they give way, and he falls to the ground. When he lies on his back and is asked to get up, he turns over with a little difficulty, and rises very much like a case of pseudo-hypertrophic paralysis. Although he does not require to place his hands on his knees in so doing, he gives the body a sudden jerk backwards in assuming the erect posture. He can bend down and touch the toes with the knees very slightly flexed, and can raise himself from this posture without using his hands. The muscularity is poor as a whole, but I cannot say that there is any definite localized wasting, nor is there any hypertrophy. The scapulae do not project, and I can detect no weakness of the upper extremities. When the patient is lifted from the ground with the hands under his arms, there is no tendency for the shoulder-girdle to move upwards. The right knee-jerk is obtained with a little difficulty, the left is absent, as are both ankle-jerks; the plantar reflexes are indefinite. There is no weakness of the face, and no defect of the ocular movements.

On March 24, 1922, the patient's doctor wrote to me as follows: "W. C. is, as far as I know, quite strong and well; it will be about a year since I have seen him, as the family have removed (from this neighbourhood for a time). He has gone through the minor children's ailments without any mishap. He is now able to ride a tricycle and a scooter; the only remains of his trouble is his gait, which has a distinct waddle."

An assertion to the effect that a case of a well-known disease which is commonly regarded as incurable has recovered demands most careful consideration before the evidence can be accepted as conclusive. The possibility of an error in diagnosis at once suggests itself as the most probable explanation of the facts, and the data must be capable of withstanding the most severe criticism before the contention can be admitted. Since Dr. Batten hesitated to give the diagnosis of pseudo-hypertrophy unreservedly when he first examined this patient, the case may appear to be of little value in relation to the question at issue: on the other hand, we have the statements of the child's mother and medical attendant that after Batten had seen the child on a second occasion he committed himself to a diagnosis of myopathy. While it is possible that the case was one of myopathy in which recovery has taken place, it seems on the facts much more probable that it was not an example of this disease, but that it so closely resembled myopathy that Batten was driven to a diagnosis by a process of exclusion. Had there been a family history of myopathy, the probabilities in favour of the diagnosis would have amounted to a certainty. This case illustrates the importance of a positive family history in any case in which the diagnosis is doubtful. Is it not possible that in the three cases of reported cure cited by Batten the diagnosis may have been incorrect, particularly since in none does there appear to have been a family history of myopathy?

## REMARKS ON MYOTONIA ATROPHICA—REPORT OF A CASE WITH AUTOPSY.

In conclusion I shall allude briefly to some features of myotonia atrophica, an affection which has been classed with the myopathies. Thus the tendency to a symmetrical distribution, the myopathic facies, the absence of fibrillary tremors and of qualitative changes in the electrical responses, the occasional presence of hypertrophy and the familial tendency, are features common to the two conditions. On the other hand, in certain respects the symptoms of myotonia atrophica differ very notably I think from the myopathies. Thus, apart from the myotonia, a symptom which has not, so far as I know, been observed in the true myopathies, the distribution of the muscular atrophy does not correspond to any known myopathy, while, further, transitional types have not been described. I would also emphasize in this relation the frequency of congenital stigmata of various kinds in myotonia atrophica, while, in addition, I have been forcibly impressed by the peculiar mental state in several cases of this disease with which I have met. Again, it is to be remembered that these patients do not die of the disease. Myotonia atrophica cannot be regarded as a disease of extreme rarity. Batten and Gibb<sup>1</sup> collected twenty-nine, and Steinert<sup>2</sup> twenty-six cases from the literature in 1909, while Addis and I<sup>3</sup> were able to find reports of sixty-one cases four years later. Twelve cases in all—ten males and two females occurring in six different families—have come under my personal observation. The symptoms may exist for long without causing any appreciable inconvenience. Thus, one of my patients, who consulted me regarding a facial neuralgia, was annoyed—an instance of the abnormal mental state to which I have referred—when I proceeded to inquire regarding his muscular development. He practically told me it was no business of mine, since he had been born like that, and had never been caused any real inconvenience thereby. Another patient who presented the characteristic symptoms of the disease in a pronounced form was sent to me with an old-standing hemiplegia which had developed in early childhood. This patient's two younger brothers whom I also examined both exhibited the myopathic facies, with some wasting of the sternomastoids and the myotonic phenomena in the upper limbs. They persisted in saying there was nothing the matter with them. All three, but notably the younger brothers, were obvious psychopaths, and their parents, who were most respectable, hard-working people, had been caused much distress by the boys' inability to realize the meaning of truth and honesty. Another man who attended my clinique was constantly changing his situations, under the impression that his employers were endeavouring to get the better of him in one way or another. At one time he flatly denied that he had any brothers, and only admitted his mistake when I confronted him with a photograph of himself and two brothers which I had taken some years previously. The testicular atrophy first noted in myotonia atrophica by Steinert in his paper published in 1909 is of interest. Addis and I found this observation recorded in eight cases. I have now seen three instances in which testicular atrophy was a striking feature. In one or two other cases, too, I was inclined to think that the consistency of the testicles was unduly soft although they were not obviously reduced in size. The testicles were about a third of their normal bulk in the case of a married man, aged 43, the father of a family of six, who has just left my ward. In this case the first manifestations of the disease had shown themselves seven years

<sup>1</sup> *Brain*, 1909, xxxii, pp. 187-205.

<sup>2</sup> *Deutsche Zeitschr. f. Nervenheilk.*, 1909, xxxvii, pp. 58-104.

<sup>3</sup> *Edin. Med. Journ.*, 1913, n.s., xi, pp. 21-44.



ago; the patient's youngest child was a year old, and for fourteen months he had lost all sexual desire. The possibility of an aetiological relationship between this testicular atrophy and myotonia arises in view of these facts. There are two other points in the symptomatology of the disease, such as the cold hands so frequently complained of, and the early loss of hair observed in some cases, which raise the possibility of a glandular disturbance as an underlying aetiological factor. The fact that testicular atrophy had been observed in almost 20 per cent. of the male cases reported up to 1913 is certainly striking. The arguments that in the remaining 80 per cent. no atrophy was observed, and that no disturbance of the primary sexual functions may be observed until the disease has been in existence for years, do not, you will probably agree, justify us in dismissing forthwith the possibility of a disturbance of testicular secretion at a much earlier date. This relationship, indeed, calls for elucidation. I may say that I have seen no obvious effects produced in two or three cases of the disease in which I have employed testicular preparations.

A pathological examination of the nervous system has only been reported, so far as I have been able to ascertain, in two cases up to the present time. One of these cases was recorded by Steinert in 1909, the other by Hitzzenberger in 1920. I shall conclude by giving you a résumé of a case which was under my care and which was examined pathologically by Dr. Dawson.

*Case VI: Case of Myotonia Atrophica with Autopsy.*—W. H., aged 52, a valet and masseur, was seen in July, 1913. On inquiry it was elicited that he was the elder brother of a family three of whom I had previously examined and found to be affected by myotonia atrophica. These cases were reported, in conjunction with W. R. Addis, in the *Edinburgh Medical Journal* for July, 1913, pp. 21-44.

*History:* The patient had been married for seven years and had one child, a girl, aged 6, who was said to be well and strong. As a young man he stated that he had been particularly strong and a first-rate athlete; he had, he told us, jumped 5 ft. 10 in.; he used to play football and he had been a good boxer. As a younger man he was able, he said, to lift two 56 lb. weights above his head. Again, he stated that he was able to stand extremely strong electric currents; thus, he said that many years previously, when at a fair in America, he and two of his companions tested themselves on an electrical instrument, when he found that he was able to stand the full strength of the current without discomfort. The showman told him that his nerves must all be dead, and offered him, he said, five dollars a day to travel round America with him for purposes of exhibition. He stated that he had never had venereal disease, and the Wassermann reactions were found to be negative. As a young man he had been a fairly heavy drinker but he stated that he had taken practically no alcohol for the past ten years.

For some sixteen years he stated that he had noticed that after firmly closing the hand he had had difficulty in extending the little and ring fingers of the right as also the little finger of the left hand. He had been unable for some years to do any heavy work because of the weakness of the grasp, but for the past six years he said that his hands had been distinctly stronger. Eight years before he came under my notice his right eye had been operated on for cataract while the left eye had been needled for the same condition six years later. Two and a half years before I saw him he had had pneumonia; he had had a cough since, and upon examination I found that he was suffering from a bronchiectasis. For three months the patient said that he had been going downhill rapidly, and that he had lost almost two stones in weight.

*State:* The facial appearance was striking; there was great hollowing in the temporal and masseter regions on either side; he often sat with his eyes half closed and the eyeballs turned upwards so that only the whites of the eyes were visible. There was no ptosis. The palate was very high and narrow. He was almost bald. The voice was distinctly hoarse; articulation was a little thick, with a distinct nasal

intention. He gave a history of occasionally having brought fluids through his nose when swallowing. He said that he had often noticed in the morning that he had difficulty in opening his eyes. The masseters and temporals were weak and much wasted. He could not close his eyes firmly and he was unable to whistle; the facial movements were indeed very defective. The sternomastoids were both distinctly atrophied; the general musculature, too, was very poor. The forearms were especially wasted, particularly on the ulnar aspect; the abductor pollicis was practically absent on both sides; otherwise there was no definite wasting of the hand muscles. No myotonic phenomena were observed in the grasp or on percussion of the other muscles though carefully tested for. (There were no fibrillary tremors nor was there any muscular hypertrophy.) Though he stated that he became rather easily tired after walking, there was no obvious localized weakness or wasting of the lower extremities. The deep reflexes were unaltered. There was no sensory loss or sphincter disturbance. The left testicle was rather smaller than the right though I could not say that it was definitely atrophied. For the past three years there had been no sexual desire. About the root of the right lung the physical signs suggested the presence of a cavity and there were some moist sounds. The sputum was tested repeatedly for tubercle with a negative result. There were two pimples, one on either side of the neck immediately below the larynx; he stated that from childhood up to eight years ago a discharge occurred from these pimples from time to time. *The patient died on March 4, 1915, the immediate cause of death being an attack of erysipelas affecting the face.*

The post-mortem was carried out by Professor Drennan on March 4, 1915; the following are some of the more striking facts observed: There was marked emaciation. The skull, membranes, brain and spinal cord showed no gross abnormality. The pituitary body was of normal size, as also was the thyroid gland. On section the thyroid was seen to be pale brown in colour, and showed no colloid to the naked eye. The left lung was smaller than the right, and the smaller bronchi, which contained thick pus, were distinctly dilated; extending from the root there were several larger bronchi, showing marked bronchiectasis; there was some recent fibrinous exudate over the lower lobe. The heart was small, the muscle of a dark brown colour. The abdominal organs showed nothing worth noting. The spleen was somewhat small and soft; the pancreas was also small; the kidneys showed a slight uniform diminution of the cortex; the suprarenals were both very thin, the medulla was small in amount, and the cortex thin and of a brown colour. The prostate was not enlarged. The left testicle appeared slightly atrophied; both testicles were soft, and on section of a light brown colour. In the situation of the thymus there was a small lobule of fatty tissue, resembling thymus in shape; in the left portion of this there was a small pocket of pus. The orbicularis oculi was very thin and pale yellow. The temporal muscles were thin and somewhat pale. The sternomastoids, which were uniformly small, were of the usual colour. In general, the change in the muscles consisted in a diminution in size; there was no evidence of fatty change, and their colour was practically normal; if anything, some muscles appeared slightly darker red than usual. Subcutaneous fat was almost absent.

The histological examination of the nervous system was carried out by Dr. James Dawson, whose conclusions were as follows: "The points to be noted in the investigation of the nervous system may be summarized thus: (1) No tabiform degeneration; no change in extra-medullary posterior or anterior nerve roots; no definite change in posterior entry zone nor in collateral fibres. No infiltration in the meninges nor walls of the blood-vessels. No uniform gross changes in cells or cell-groups of grey matter nor posterior root ganglia (except pigmentation); no changes in the nerves of the brachial plexus nor cauda equina. (2) Weigert and Marchi sections show only the slightest evidence of degeneration; there is marked pigmentation of the anterior horn cells and cells of the posterior root ganglia; and amongst these cells are very numerous amyloid bodies; in the column of Burdach, at level of the third cervical segment, occur a few heterotopic ganglion cells."

(The various tissues which were kept for histological examination, with the exception of the spinal cord which was handed over at the time to Dr. Dawson, have unfortunately been mislaid.)



## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### Multiple Peripheral Neuritis :

#### PRESIDENT'S ADDRESS.

By WILFRED HARRIS, M.D.

RECOGNITION of multiple or peripheral neuritis as a clinical and pathological entity dates from the publications of Duménil in 1864, and especially of Leyden in 1878 and later. Since that time a host of observers have studied the disease, so that the varieties of the commoner forms, the alcoholic, lead, arsenical and diphtheritic paralysis are well known. It is not my intention this evening to dwell on those forms the ætiology of which is clear and unmistakable, but rather on the numerous lesser known types the origin of many of which is quite obscure. Multiple neuritis in our present state of knowledge is best classified on an ætiological basis rather than on pathological distinctions or clinical differences, and four main groups may be separated: (1) Those dependent on the absorption of poisons introduced into the body; (2) those due to autotoxæmia, or poisons developed primarily within the body; (3) infective causes, of an organismal nature; (4) cachectic.

The first group may usefully be divided into two sub-groups, those due to metallic poisoning, and those due to poisoning by various carbon compounds.

The term "toxic polyneuritis" has been much used, but, as probably all polyneuritis is dependent on some toxin, its employment seems most appropriate for those cases in which the infective agent or the source of poisoning is not clearly established. Before dealing with these a few examples of rarer forms from the different groups may be referred to.

Gowers<sup>1</sup> used to tell a story of a man who consulted his doctor brother for some gastric disorder, and was given a dinner pill containing a small quantity of silver oxide. For about twelve years he took about six pills a month, and after eleven years he noticed that his lips and face appeared slightly bluish, and so he again consulted his brother, who sounded his heart carefully, and then told him not to worry. Nothing was said about the dinner pill, which he continued to take as before, until wrist-drop developed, for which Gowers was consulted. Argyria is a very rare disease in these days, as silver is now used practically only in lotions. There seems to be no other case of silver polyneuritis in the literature, and, as Gowers' patient died three months later of carcinoma of the liver, it may possibly have been a polyneuritis secondary to malignant disease and not due to silver.

<sup>1</sup> "Diseases of the Nervous System," ii, 2nd ed., p. 967.

As a complement to the case just described, I might perhaps refer to a patient of mine who was saved from having a gastro-jejunostomy operation performed on him for the relief of supposed duodenal ulcer, by his butler developing on the day previous to that fixed for the operation a pronounced wrist-drop. Investigation then directed to the water supply showed that the piping, bringing the supply to the house some distance from the main in the road, had been laid with lead pipes instead of iron, as contracted for.

*Carbon bisulphide* is sufficiently uncommon as a cause of polyneuritis to justify a short description of a case I have had recently under treatment.

A boy, aged 15, worked at a boot factory at solutioning on rubber soles. He worked in a shed, which was afterwards described by the inspector as very badly ventilated, using for the purpose of fastening on the rubber soles a rubber solution containing carbon bisulphide. He worked from 8 a.m. to 6 p.m., and began to feel unwell in July, 1921, when he began to suffer from headaches almost every night towards the end of his work. The headaches were accompanied by vertigo, but had disappeared by the following morning. He informed me that previous workers at the same job had had to give it up on account of similar headaches and giddiness. He, however, stuck to his work, and in January of this year his right leg began to feel weak and to give way when walking towards the end of the day. This improved, and then the right hand grew weak and lost the power of gripping; this also improved, and the left hand became similarly affected. The headaches ceased in January, just before the legs began to be affected. There was never at any time any pain or numbness or any sensory symptoms, objective or subjective. By the beginning of April he began to develop weakness of the dorsiflexors of both feet, being unable to raise the toes off the ground when standing, though the gastrocnemii were strong. Soon also bilateral weakness of the extensors of the fingers and wrists appeared, especially of the right extensor carpi ulnaris. Marked weakness and wasting also of the right first dorsal interosseous and thenar muscles was seen. To electrical testing there was well marked reaction of degeneration in the extensors in both forearms, in the right first dorsal interosseous and the tibialis anticus on each side. The knee-jerks were normal throughout, but the right Achilles-jerk was absent, and the left only just obtainable with reinforcement. Treated with strychnine injections and massage he soon showed slight improvement, and after four months had practically recovered.

*Carbon monoxide* polyneuritis I have never met with, but it is said that a single severe dose of poisoning with this agent may be followed by neuritis; a very unusual feature in polyneuritis, which is usually the sequel of a more sustained or chronic poisoning. Care must however be taken in cases of carbon-monoxide poisoning not to mistake pressure neuritis of the musculo-spiral, external popliteal or other nerve trunks for toxæmic neuritis; pressure palsies being especially liable to occur in persons rendered unconscious by coal-gas poisoning or in explosions in coal pits, through lying on a hard surface in one position for many hours. I have, however, seen a most severe attack of multiple neuritis follow immediately after an *intense chill*, in a woman whose bed was soaked through by the rain breaking through the roof. No alcoholism or other cause than the chill could be discovered in this case, which was labelled *rheumatic polyneuritis*.

Chloretone has been given extensively to children for chorea, and has been observed to produce a condition of flaccid paralysis, with absent deep reflexes, but the recovery on cessation of the drug is usually much more rapid than is usually met with in other forms of polyneuritis.

Acute rheumatism or rheumatic fever is rarely if ever followed by polyneuritis, yet local perineuritis and fibrositis, as seen in brachial neuritis and

sciatica, are common sequelæ of chill and exposure, though other causes frequently contribute, such as dental or intestinal sepsis.

Local *asymmetrical neuritis* of definite nerve trunks rather than a symmetrical polyneuritis is also a feature of typhoid and paratyphoid infections. Thus a man, aged 29, with paratyphoid, a week after admission to hospital, developed severe pains in the upper arms and shoulders, which kept him awake, and were followed by marked paresis of the right serratus, latissimus and triceps, the extensors of the right fingers and thumb, and the left deltoid.

Another case of asymmetrical severe neuritis of the right median, ulnar and musculo-spiral nerves occurred in a man, aged 27, as the sequel of a febrile attack of uncertain origin, with tonsillitis, followed by acute nephritis. There was never any peeling of the skin to suggest scarlet fever, and bacteriological examinations for diphtheria, enteric and paratyphoid were negative. Possibly the infection was rheumatic, as neuritis is one of the rarer complications of this disease. After two weeks he had severe pains in the right arm from the shoulder to the hand, completely losing the use of the arm, with anæsthesia of the hand up to the wrist. The hand became very clawed and atrophic, with atrophy of the finger pads and skin, though practically complete recovery took place in two years.

Asymmetrical neuritis of toxic origin may occasionally be met with in chronic alcoholism and diabetes. Musculo-spiral paralysis I have many times seen occur in chronic alcoholism, when the element of pressure could apparently be excluded as a cause. In none of these cases of musculo-spiral palsy was pain a feature of the case, but in the sciatic and external popliteal paralyses met with in diabetes, severe pain may be a prominent feature preceding and accompanying the paralysis. It is difficult to understand how a local asymmetrical neuritis can be set up by the action of a circulating toxin, which should presumably act equally and symmetrically on both sides. The fact that definite whole nerve-trunk areas are liable to be affected in the asymmetrical forms is suggestive of a local interstitial neuritis being set up rather than a degenerative parenchymatous neuritis, and this is further supported in my opinion by the fact that these asymmetrical forms are usually intensely painful. It is possible, therefore, if not indeed probable, that the circulating toxin is not the direct factor in the development of the neuritis, but that it has led to the local appearance of an inflammatory interstitial neuritis, such as may result from chill and other so-called rheumatic processes.

The actual nature of the circulating toxin in such apparently simple cases as alcoholic or diabetic multiple neuritis is really most obscure. In all probability it is not the actual alcohol or derivative ethers circulating in the blood that sets up alcoholic neuritis, or this disease would surely be commoner than it is, but rather a secondary auto-toxæmia produced by the action of the chronic long-sustained alcoholism upon the tissues, notably of the digestive tract and the liver. Similarly in diabetes it can scarcely be the direct action of hyperglycæmia on the nervous structures that sets up local or multiple neuritis, nor, certainly, is it due to acetone or diacetic acid, as both of these substances may be absent in cases of diabetic neuritis. Probably the neurotoxin in diabetes is of the autotoxæmic deficiency type; this will be referred to later.

Alcoholic neuritis has become much rarer in this country since the restrictions imposed by war conditions, and owing to prohibition it has become a rarity in America.

The *polyneuritic psychosis*, described by Korsakow<sup>1</sup> as common in chronic alcoholism with polyneuritis, is certainly met with in other forms of polyneuritis, as Korsakow himself showed, and must therefore not be accepted as evidence proving alcoholism to be the cause of an obscure polyneuritis. Some two years ago I had the opportunity of watching throughout to its fatal ending a case of polyneuritis of unknown origin, in which the delusions and characteristic disorientation to time and space, familiar in Korsakow's psychosis, were very marked.

A man, aged 55, hall porter, early in September, 1920, began to suffer from severe pains in the left hip. After a fortnight he had to give up his work, and three weeks later pains appeared in the right hip. On October 16 he was admitted under my care in St. Mary's Hospital as a supposed case of sciatica. There was some loss of power in both legs, and tactile anesthesia of the feet and ankles, but no loss of power or of sensation in the arms or hands. Both knee-jerks and Achilles-jerks were absent; the plantars flexor. No glycosuria or albuminuria. Wassermann test of blood and cerebrospinal fluid both negative. On October 26 the electrical reactions were normal, but on November 5 definite reaction of degeneration had appeared in the left dorsiflexors and the peronei of both legs. By November 12 bilateral foot-drop was well marked, and there was retention of urine, daily use of a catheter being necessary. His mental condition had gradually become unreliable, and by this date delusions were very marked; he muttered to himself, and would reply in a most extravagant manner to questions as to where he had been recently, as "to New York yesterday," and "to Jerusalem the day previously," though he would otherwise answer questions quite rationally. He had always consistently denied the imputation of alcoholism, and I was able to convince myself by personal interviews with his wife and with his fellow employees at the town hall where he was porter, that his statements on this point were true. Irregular pyrexia developed towards the end of October, and he steadily got weaker and more torpid, dying on December 14. No post-mortem was permitted.

In another case, a man aged 45 had a severe attack of polyneuritis, with intense pains in the feet and legs, with loss of the deep reflexes, and considerable delirium. Alcohol as a cause could be excluded. He later recovered all his muscular power, and the knee-jerks returned, but there was permanent and considerable loss of memory and mental power, indicating the co-existence of an acute encephalitis with the polyneuritis.

*Septicæmia* is a not uncommon cause of multiple neuritis differing little in type from alcoholic neuritis. The source of infection may be quite an insignificant wound; in two cases I saw well marked multiple neuritis follow slight local suppuration due to a grazed skin-wound of the hand, and in another case due to a stab wound in the thigh. In one of them an early symptom was loss of accommodation power, which is such a common early symptom in post-diphtheritic neuritis.

Septicæmia, again, is a not uncommon cause of multiple neuritis following child-birth or a miscarriage; one of the most severe and painful cases I have ever seen, ending in death from heart failure, occurred in an otherwise healthy girl aged 23, after a miscarriage at three months, in all probability an artificially induced abortion. The polyneuritis which has been described as following tetanus is almost certainly of septicæmic origin, rather than due to tetanus toxin, though chloretone used in the treatment has been blamed in one case. The use of antistreptococcic serum I have seen followed by a severe external popliteal paralysis.

A gunshot wound necessitated amputation of the foot, but on account of sepsis three doses of antistreptococcic serum were given on three successive days, and then

<sup>1</sup> *Arch. f. Psych.* 1890, xxi, p. 669.

after four days' interval another dose. General joint pains all over resulted, followed four days after the last injection by pains in the right leg, dropped foot and wasting of the leg, with total paralysis of the extensor longus digitorum and of the peronei. Was this a septicæmic neuritis, or was it the effect of the serum?

Septicæmic neuritis may affect a nerve trunk close to the intervertebral foramina, as in a case of a gunshot wound of the left arm, followed four weeks later by pains in the opposite arm and right side of the neck, with paresis of the right serratus magnus and winged scapula, and hypo-æsthesia of a cutaneous strip from the ball of the thumb along the outer forearm and up to the shoulder, suggestive of neuritis of the sixth cervical nerve.

Generalized polyneuritis affecting all four limbs, with ataxy and loss of the knee-jerks, has been recorded by Clayton in a soldier treated for typhoid by fifteen injections of 50 c.c. each of antityphoid serum. Here again it may be asked whether the polyneuritis was the result of the serum injections, or whether it was due to the typhoid toxin. My own view is that the neuritis is not the result of the serum treatment, but of the previous toxæmia, just as in cases of polyneuritis following diphtheria, whether treated with serum or not, the neuritis should certainly be laid to the credit of the diphtheritic toxin.

A rare form of polyneuritis has been described by Feiling<sup>1</sup> and others accompanying *parotitis* and *uveitis*, or polyneuritis may follow parotitis alone.

*Acute febrile polyneuritis*, first described by Osler,<sup>2</sup> was frequently observed during the Great War, and was described by Gordon Holmes,<sup>3</sup> Bradford and others.<sup>4</sup> The sequence of the polyneuritis to definite malaise and pyrexia places this type definitely in the infective rather than in the autotoxæmic group, though the exact source of the toxin has not yet been worked out. Facial diplegia was a common symptom in this type of polyneuritis.

I saw a medical officer, just home from France, early in 1919, who complained of pains in the left side of the neck, with soreness to touch, as though bruised; almost immediately aching appeared in both forearms; three days later insomnia, and the arms became weak and the hands shaky. After three weeks, facial diplegia developed, with a feeling of intense depression and malaise, with furred tongue, though no pyrexia was detected at any time. There was a good deal of pain with hyperæsthesia and tenderness of the backs of the forearms, but no actual muscular paralysis. The knee-jerks were present throughout, and the legs were unaffected. There was never any sensory loss, and recovery was complete after three months.

*Syphilis* as a cause of polyneuritis is usually included in the cachectic group, but occasionally it undoubtedly acts as an acute infective toxæmia.

A man, aged 30, acquired syphilis in January, 1910. Three months later he developed some ataxy and numbness of the feet, but recovered. In October the numbness and ataxy returned with rapid onset of weakness of legs and arms, but again he almost completely recovered under injections of salicylate of mercury. In January, 1911, twelve months after the original infection, weakness and wasting of the hands and feet began, which steadily progressed to complete paralysis of all muscles below the knees, with bilateral finger-drop and wasting of the forearm muscles. All the deep reflexes were lost, but there was no trace of anæsthesia. By November, 1912, he was recovering fair power in the gastrocnemii, and he could walk fairly with a stick. The hands were now normal, and the knee-jerks had returned, but there was

<sup>1</sup> *Journ. of Neur. and Psych.*, 1922, ii, p. 353.

<sup>2</sup> "Principles and Practice of Medicine," 1892, New York and Lond., p. 777.

<sup>3</sup> *Brit. Med. Journ.*, 1917, ii, p. 37.

<sup>4</sup> *Quart. Journ. Med.*, 1918-19, xii, p. 88.

no trace of power in any of the anterior tibial muscles, nor any reaction in them to either form of electrical current. Probably the correct interpretation of this case would be a polyneuritis associated with considerable anterior horn cell damage in the fifth lumbar region.

In considering the ætiology of an attack of multiple neuritis in a syphilitic subject, the possibility of arsenical neuritis should not be overlooked if treatment by arseno-benzol preparations has been given.

*Tubercle* as a cause of polyneuritis has been much discussed, the usual verdict being that such polyneuritis is mainly alcoholic, though the added stress of the tuberculous toxin or cachexia, when combined with the chronic alcoholism, much increases the liability to polyneuritis, just as alcohol plus arsenic, sepsis plus rheumatism, and many other doubly acting causes, may combine to produce polyneuritis. Apart from such doubly acting causes, tubercle is a very rare cause of neuritis. I have seen one case of generalized polyneuritis in a man dying from miliary tuberculosis, with no history of alcoholism or other ascertainable cause of neuritis.

*Tuberculous Neuritis of the Feet.*—In two cases of severe and fatal tuberculous infection, I have seen symmetrical pains and wasting of the intrinsic musculature of the feet, with glossy skin. The first case had been diagnosed as gout, in spite of extensive pulmonary tuberculosis, by no less an observer than the late Sir William Broadbent in 1895, and remembering this case when I saw the identical condition of the feet seven years later in a woman with obscure renal disease and pyrexia, I confidently diagnosed tubercle as the cause, though no one else agreed with me until the autopsy confirmed this.

In a few cases I have seen tuberculous pleurisy followed by intensely severe *brachial neuritis* on the same side, with glossy skin and articular disorganization of the phalanges. Probably this is produced by apical infection of the lung and pleura setting up a secondary inflammatory neuritis in the brachial plexus in its close proximity.

Amongst the *cachectic* causes of polyneuritis, severe chronic anæmia is usually quoted as a cause, though it appears to be much rarer than the spinal degenerations of combined sclerosis met with in conjunction with grave anæmia of pernicious type.

I published last year<sup>1</sup> a case of leukæmic polyneuritis in a youth, aged 17, who developed well marked symmetrical polyneuritis, with foot-drop and absent deep reflexes and sensory diminution on the feet and legs, with tingling and numbness of the finger pads. His spleen was enlarged three finger-breadths, but there was no leucocytosis; indeed there was a leucopenia of only 5,800, but the cell count was notably inverted, the polymorphs being only 23 per cent. and the lymphocytes 68 per cent. Slight enlargement of the glands in the neck appeared, and one of these was removed for pathological examination, but was reported to be normal. Troublesome hæmorrhage from the wound continued at intervals, again suggestive of leukemia, and he died six weeks after admission into St. Mary's, three months after the first appearance of weakness of the legs.

Microscopical examination of the liver, kidneys and peripheral nerves showed these tissues to be infiltrated with lymphocytes; yet it is difficult to conceive that the symmetrical polyneuritis could be due to pressure of the lymphocytic exudation upon the nerve fibres. It must rather have been of toxic origin due to some unknown form of autotoxæmia connected with the leukæmic process.

<sup>1</sup> *Lancet*, January 15, 1921, p. 122.



*Malignant disease* is also included in the cachectic group of polyneuritis, though it seems to be rare.

A year ago I saw a civil engineer, aged 69, who had been operated on seven years previously for malignant growth of the ascending colon, a short circuit having been done. He recovered well, and for years lived an ordinary life, travelling a great deal. In October, 1921, he thought he caught a chill, followed by pains in the left shoulder, arm and forearm, "like a rat gnawing it." The pains spread across the back of the neck, and then affected the right forearm, being so severe as to necessitate the administration of morphia. When I saw him in December there was complete paralysis of both triceps, and of the extensors of the fingers and ulnar extensors of both wrists; the radial extensors of the wrists and the supinator longus of each side were normal, though both serratus magnus muscles were very weak. There was a large mass in the right hypochondrium, which was shown at operation to be a growth in the ascending colon, with most extensive involvement of the liver. The legs were quite normal, and the knee-jerks brisk; there was no glycosuria.

The distribution of the polyneuritis in this case was most unusual, the legs being quite unaffected, with bilateral finger-drop and total paralysis of both triceps muscles. The intensity and long duration of the pain were also a striking feature of the case. Possibly chill contributed to the onset of the neuritis, but the long duration of three months with increasing symptoms were more suggestive of a continually-acting toxin or cachexia, and is better explained by the extensive carcinoma from which he was suffering.

*Autotoxæmia*.—Perhaps the most interesting group of causes of polyneuritis is that which I have labelled "autotoxæmic." Constantly we are meeting with cases in which the closest investigation can elucidate no cause for the polyneuritis. One is tempted therefore to look upon them as autotoxæmic, though the Scotch verdict of "not proven" would be more correct.

An unmarried woman, aged 47, a school teacher, early last November (1921) began to suffer from burning pains in the legs, the feet becoming numb two months later, with loss of power of walking. There was some swelling of the hands due to rheumatoid polyarthritis, which also involved the left knee-joint. She complained of pins and needles in the fingers, finger-drop from paresis of the extensor communis, and weakness and wasting of the intrinsic musculature of the hands. There was tactile anæsthesia and analgesia of the left hand up to the wrist, but no objective loss of sensation on the right hand, though she said it did not feel natural. Complete left foot-drop and some weakness of the right dorsiflexors were present. She could not move the left toes, but could move the right toes. Though the right foot was much stronger than the left, yet there was much denser anæsthesia of the right foot and leg, extending to just above the knee. The knee-jerks were both brisk and equal, though the Achilles-jerks were absent, the left forearm flexor-jerk being absent, while the right was brisk. After two months the pains in the legs increased considerably, and she complained of a curious subjective sensation "as though she had several pairs of legs in bed." This continued for several weeks. Now after eleven months there is some improvement in the muscular power of the legs, but she is still only just able to walk. There was a perforation of the nasal septum, suggestive in appearance of old syphilis, probably congenital, but the blood Wassermann was negative.

It was difficult to assign the cause of this polyneuritis, though I incline to look on it as *rheumatic polyneuritis*, in view of the presence of multiple joint swellings, with pain, and the asymmetry and painfulness of the polyneuritis.

*Beri-beri* was at one time considered to be an infective polyneuritis due to a bacterial poison, but the more recent view ascribes it to a deficiency of



vitamins in the diet, due to the eating of polished rice; the husk of the grain, which is thus lost to the diet, containing all the vitamins. Such a deficiency cause must, of course, act indirectly only, by setting up changes in the metabolic processes of the tissues and thus probably producing some insufficiently broken down excretory product which in the course of circulation poisons the peripheral nerves, causing symmetrical parenchymatous degeneration. The process may be compared to that in *diabetes*, in which the lack of the normal internal secretion of the cells in the islands of Langerhans in the pancreas so alters normal metabolism that an excess of dextrose is produced in the breaking down of the muscular tissue. Yet it is probably neither the dextrose in the blood nor the acetone or diacetic acid by-products which are the actual toxic agent in producing the neuritis. In diabetic neuritis there may be symmetrical numbness and anaesthesia of the extremities with loss of deep reflexes and with little or no muscular paresis but well marked ataxy, the so-called diabetic pseudo-tabes. In another form the pains may be extremely severe, and the muscular paralysis well marked, often in the distribution of one sciatic, external popliteal or anterior crural nerve. Why a circulating toxin should thus set up an asymmetrical neuritis is hard to understand; it is usual to explain this by inferring a locally acting cause in addition, such as overuse or strain of the limb, or undue pressure on the nerve. In the asymmetrical neuritis of rheumatism or of paratyphoid, the asymmetry may be due to local involvement of the nerve sheaths by direct organismal infection, somewhat after the same manner as leprous neuritis, but in diabetes no organisms are known to be at work.

*Puerperal Neuritis.*—Polyneuritis developing shortly after childbirth or even during the puerperium, is sometimes considered to be a septicæmic neuritis, due to sepsis acquired during parturition, or to retained septic products in the uterus from a previous pregnancy. I do not myself think that this explanation will cover all the cases of polyneuritis associated with pregnancy, but rather that some are true cases of autotoxæmia, in which a toxin which produces the polyneuritis is elaborated in the tissues as an abnormal by-product of the pregnancy metabolism.

A year ago I had under my care for some months a young married woman, aged 30, who ten weeks after childbirth complained of weakness of the legs and of numbness and pins and needles spreading in the feet and hands, followed by numbness of the abdomen and chest up to the neck. All the deep reflexes were lost, and she was unable to lift the legs off the bed, while the extensors of the fingers also became weak, but the extensors of the thumb and of the wrist were unaffected. Bilateral facial palsy next appeared and diplopia, followed by diaphragmatic palsy for several days.

There was deep tenderness of the calves and forearms, and well-marked anaesthesia of the feet and ankles, but she never suffered from any spontaneous pains. There was never pyrexia or recognizable sepsis after the childbirth. She recovered completely after four months.

Ten years ago the late Dr. F. E. Batten showed at a clinical meeting of this Section a woman with recurrent paralysis and wasting of the hands and legs, with absence of the deep reflexes, the paralysis recurring after two confinements. Such recurrent paralysis will be referred to presently as having been observed in other cases of apparently autotoxic polyneuritis.

*Pressure neuritis* as a cause of paralysis following childbirth must be carefully distinguished from toxic polyneuritis, the nerve that usually suffers being the sciatic, and especially its external popliteal portion, from pressure of the foetal head upon the nerve near its exit from the pelvis at the sciatic notch.

Indeed, so vague are some medical men in dealing with "nerve" cases, that I saw one such case of left sciatic palsy following immediately after childbirth—complicated by musculo-spiral palsy of the right arm from pressure on the edge of the bed during the anæsthetic, as forceps had been used—which had been diagnosed as a case of "crossed paralysis," whatever that may have been supposed to mean.

*Cranial nerve paralyses* are common as a sequel of ordinary pharyngeal diphtheria, the third, fourth and sixth nerves being commonly affected, and occasionally the seventh, fifth, tenth and twelfth. The loss of accommodation that is such an early sign in post-diphtheritic palsy, due to paresis of the ciliary muscle, I have also seen in septicæmic neuritis and in one other case of toxic polyneuritis. Walshe<sup>1</sup> has pointed out that the frequency of incidence of cranial nerve paralyses after diphtheria is dependent on the close contiguity of the source of the toxin in the pharynx, as cranial nerve paralyses are uncommon in cases of polyneuritis following accidental diphtheritic infection of an open wound of the trunk or limbs.

A definite syndrome has been claimed for *polyneuritis cranialis*, several observations being recorded of neuritis limited to several cranial nerves. Oppenheim<sup>2</sup> thinks these cases are usually due to a basal process involving the cranial nerves. Thus Kinnier Wilson<sup>3</sup> showed at the Clinical Section (February 13, 1920), a man who, after three weeks' right-sided otitis media, developed right nerve deafness, right facial palsy, paralysis of the right half of the soft palate and vocal cord, glossopharyngeal anæsthesia and hemiatrophy of the right half of the tongue, with bilateral loss of taste.

In a few cases of polyneuritis *optic neuritis* has been met with, as in the puerperal form, and I have seen it in a man who slowly developed foot-drop with anæsthesia of the feet and hands, pains, wasting of the anterior tibial muscles, and loss of the deep reflexes, a sequel of syphilis twenty years before, the blood Wassermann reaction being positive. No other sign of tabes was present.

*Slow chronic polyneuritis* is a term that may be applied to cases in which the symptoms gradually increase during many months. Pain is usually either absent throughout, or only trifling at the commencement, but the muscular paralysis becomes extreme, and there may be extensive atrophy.

In one case I saw, in a man aged 31, the symptoms gradually increased during twelve months, commencing with pain in the calves and a tired feeling, after the appearance of some large purpuric blotches on the legs. The legs gradually became paralysed to the stage of total flaccid palsy, the hands and forearms not becoming involved until nine months after the commencement of symptoms. When I saw him seventeen months after the onset, there was practically complete palsy of the feet and legs, hands and wrists, with contracture of the feet and hands. All the deep reflexes were lost and there was considerable anæsthesia of the periphery of all four limbs. The sphincters were normal and no cause suggestive of polyneuritis could be discovered. Wassermann test negative in both blood and cerebro-spinal fluid.

A girl, aged 15, became gradually weaker during twelve months until all four limbs were totally paralysed; she could not raise the head from the pillow; the eyelids and lips were partially paralysed; there was weakness of the masticatory muscles and the tongue was somewhat wasted. No sensory symptoms were present at any time,

<sup>1</sup> *Brain*, 1920, xliii, p. 74.

<sup>2</sup> "Text-book of Nervous Diseases," Edin., 1911, p. 515.

<sup>3</sup> *Proceedings*, 1919-20, xiii (Clin. Sect.), p. 65.

except slight paræsthesiæ of the fingers at the commencement. All the deep reflexes were lost, but only slight muscular wasting occurred, and no contractures. No improvement was observable in spite of daily massage and electrical treatment until eighteen months after the onset, when she slowly began to regain power, and ultimately recovered perfectly in another two years.

The slow development of the symptoms in such cases strongly suggests a continuously acting toxin, rather than an acute poisoning, and one is driven to the supposition that an autotoxæmia is at work. In the girl's case, at least, the continual absorption of external toxins could be excluded, as her first symptoms occurred when she was at school in South Africa, and continued for three months at home some distance away, and she continued to get weaker for another nine months after being sent to England.

In sharp contrast to this slow development may be compared the case of a boy, aged 5½, who rapidly developed paralysis of all four limbs within six days, being unable to stand on the third day. Some flexor contracture developed and some muscular tenderness, but no other sensory loss. All the deep reflexes were lost early. Complete recovery occurred in nine months. No history of sore throat or any other known cause of polyneuritis could be elicited, the only suspicious point being that he had had a bad cold six weeks before. The acuteness of the onset of the polyneuritis is suggestive of an external infection being responsible, though it may be too late when polyneuritis first shows itself six weeks afterwards to detect the original infection. Acuteness of onset of the polyneuritis is not however sufficient reason to exclude an autotoxæmic cause, as will be seen presently when dealing with acute hæmatoporphyria.

*Relapsing and Recurrent Polyneuritis.*—The commonest causes of recurrent polyneuritis are chronic alcoholism and lead poisoning, but recurrence is also met with rarely in cases in which the cause is obscure.

F. G. Thomson in 1910<sup>1</sup> recorded the case of a youth, aged 19, whose weakness commenced in the hands and feet five days after being thrown out of a sculling boat, and within another ten days there was complete paralysis of all four limbs and of both sides of the face, with considerable weakness of the trunk muscles. The sensory symptoms were confined to slight numbness of the fingers and the soles of the feet. All the deep reflexes were lost, and considerable wasting developed later in the muscles about the shoulders, forearms and legs, the extensors being chiefly affected. Recovery was complete in every respect in about two years.

An interesting fact in the history was that this boy at the age of 5 had had apparently a similar attack of motor paralysis affecting all four limbs, ending in complete recovery in eighteen months. The acuteness of the onset of the paralysis following the immersion is strongly suggestive of chill being the exciting factor, though his previous similar attack at the age of 5 seems to demonstrate a predisposition in his metabolic processes to the development of a neurotoxin, or possibly an excessive sensitiveness of his peripheral nerves to the action of such neurotoxin.

In 1898 H. M. Thomas published the case of a man, aged 28, who had had five attacks of polyneuritis in successive years, and a somewhat similar case was published in 1921, by Natrass,<sup>2</sup> of a boy, aged 18, with double foot-drop, loss of deep reflexes, and other signs of generalized multiple neuritis, who had had two similar attacks of paralysis, lasting for several months, one

<sup>1</sup> *Brit. Med. Journ.*, 1910, ii, p. 1443.

<sup>2</sup> *Journ. of Neur. and Psychopath.*, 1921, p. 159.

at the age of 4 and the other at 17. A peculiarity in his case was enlargement and tenderness of the nerve trunks, such as the median, ulnar and external popliteal.

*Hæmatoporphyrinuric Polyneuritis.*—In 1908 my attention was first drawn to this curious syndrome; a woman, aged 47, being admitted to St. Mary's Hospital with pains in the abdomen, following a cold three weeks previously. Her pulse was 120, but there was no pyrexia. The urine was noticed to be dark reddish coloured, and hæmatoporphyrinuria was demonstrated on analysis. A week later she began to complain of pains all over her limbs and body, like knives, and it was found that she could not raise the arms to the level of the shoulder. The pains were worst in the arms and shoulders, but no anaesthesia was found. In five days she was unable to sit up or to feed herself. Insomnia became troublesome, and there was retention of urine, with overflow. The pains in her arms became so severe that she complained of feeling as if the skin were being torn off. The knee-jerks were present on admission, but were afterwards lost for some weeks. Three months later she could just hold out her arms, but there was still complete paralysis of the extensors of the fingers on both sides, with reaction of degeneration, though the extensors of the wrist and the supinator longus were good. Ultimately she recovered completely, the hæmatoporphyrinuria having disappeared before she left hospital.

It was not until February, 1921, that I met with another case of this disease. A man, aged 45, well nourished and living in perfect conditions in the country, when apparently in perfect health, except that he had always been obstinately constipated, was seized with pains in the back, thought to be rheumatism. Next day his urine was dark red coloured, the pulse was 60, and he now complained of severe pains around the loins and the front of the abdomen, at first thought by his doctor to be renal colic, and then an acute abdomen, for which he was sent into a nursing home, and a surgeon was fetched from London. The surgeon, however, refused to operate, making a diagnosis of acute pancreatitis. All the next week his pains were severe, and he was said by his doctor to be jaundiced, with bile in the urine. On the fourth day there was retention of urine, requiring the use of a catheter. After eleven days he became excitable and nervous, for which I was asked to see him. The pulse was then 100, the strength of the limbs and trunk was good, and he could stand and walk. The knee-jerks were then normal, as were all the other reflexes. He complained of some paræsthesiæ of the trunk, and, taken together with his excitable nervous condition and sleeplessness, I thought he was developing acute mania. There was no history of venereal disease, and I found the cerebro-spinal fluid normal. Two days later I saw him again; he could still stand, but the knee-jerks were now absent, though there was no weakness nor anaesthesia. He again had retention of urine, and I witnessed the doctor pass the catheter, and was astonished to see that the so-called bile-stained urine was port-wine coloured—an extreme example of hæmatoporphyrinuria, which was confirmed soon after by spectroscopic analysis. Three days later he complained of "deadness" of the chest and abdomen when touched, and I found large areas in this region now analgesic. Paresis of the deltoids was now well marked, as well as weakness of the flexors of the hips, but no other weakness of the limbs. His rectal temperature was now for the first time slightly raised, and two days later his temperature was 100° F. and the pulse 132. He was now much weaker, unable to turn in bed, and unable to cough owing to weakness of the abdominal muscles. The constipation became extreme, the diaphragm became paralysed, and he died after an illness of three weeks' duration. No autopsy was permitted.

Needless to say, in both these cases sulphonal and other drugs as a possible source of poisoning were excluded. In both cases acute abdominal pains were amongst the earliest symptoms, followed immediately by hæmatoporphyrinuria, rapid onset of pains all over, with paræsthesiæ and paralysis, especially of the arms, loss of knee-jerks, retention of urine, and insomnia. In neither of my two cases was vomiting remarked.

*Hæmatoporphyrinuria* may occur with symptoms of acute illness quite unconnected with the taking of any drug, such as sulphonal, trional, veronal or its derivatives. Acute hæmatoporphyrinuria is ushered in by severe pains in the abdomen and loins, constipation, and sometimes vomiting. Dark brown to port wine coloured urine is an early symptom, and the pains continue and may spread all over the trunk and limbs. The pulse-rate is raised to 100 or 120, but usually there is no pyrexia. Retention of urine and even incontinence may occur, and occasionally mental symptoms may be noticeable, such as general nervousness, excitability, or even delirium, and epileptic fits. In Günther's excellent article published in 1911,<sup>1</sup> there were quoted only four cases to be found in the literature, in which severe paralysis rapidly developed, followed by death soon after the onset of the paralysis, the earliest recorded case being that of Ranking and Pardington in 1890.<sup>2</sup> They had two patients affected with hæmatoporphyrinuria in the same house at the same time, both neurotic and dyspeptic, with hypogastric pain, and suppression of urine. Their first and fatal case was given 20 gr. of acetanilide a few hours before the hæmatoporphyrinuria first appeared, though Günther omits to mention this point. Grund in 1919<sup>3</sup> referred to other cases, including his own, in which symmetrical multiple neuritis lasted long enough for bilateral wrist-drop and muscular wasting, with reaction of degeneration, to be observed before the death or recovery of the patient. Amongst these cases is that of Schulte,<sup>4</sup> who described the case of a ship's cook at Kiel, aged 35, who was seized with abdominal pains and hæmatoporphyrinuria, followed by typical multiple neuritis affecting the arms only, paresis of the deltoids, biceps, supinator longus, infraspinatus and extensors in the forearms. The pulse was only 48, there was no pyrexia, and the man recovered.

Bachlechner in 1914 (Diss. Erlangen) described a case of hæmatoporphyrinuria followed by symmetrical wrist-drop and foot-drop, with reaction of degeneration, in which the paresis developed, with two attacks of the acute symptoms, and Günther in 1920<sup>5</sup> states that repeated attacks of the syndrome of acute hæmatoporphyrinuria may occur within a year, though in none of his cases did polyneuritis develop. Langdon Brown and Williams had previously in 1909<sup>6</sup> described a case with three recurrent attacks, in which epileptic fits occurred, but no paralysis.

L. F. Barker and Estes in 1912,<sup>7</sup> in an article on "Family Hæmatoporphyrinuria," describe the case of a girl of 18 with hæmatoporphyrinuria and polyneuritis, ending fatally after sixteen months, passing dark urine for the whole of that time, and suffering from pain in the lower abdomen, recurring nausea and vomiting, vertigo and epileptic fits. Laparotomy disclosed a dilated pylorus and duodenum. Two months later, after complaining of severe pains all over, bilateral wrist and foot-drop developed, with extensive atrophy, including the intrinsic hand muscles. The deep reflexes became lost, after being exaggerated, and she died four months later. At the autopsy great distension of the stomach and duodenum was found.

Unfortunately these authors give no detailed account of the autopsy, such as of the nervous system, promising a full account in a later paper, which, again unfortunately, has never been published.

<sup>1</sup> *Deutsch. Archiv f. Klin. Med.*, 1911-12, cv, pp. 89-146.

<sup>2</sup> *Lancet*, 1890, ii, p. 607.

<sup>3</sup> *Zentralb. f. Inn. Med.*, xl, p. 810.

<sup>4</sup> *Deutsch. Archiv f. Klin. Med.*, 1897, lviii, p. 313.

<sup>5</sup> *Deutsch. Arch. f. Klin. Med.*, 1920, cxxiv, pp. 257-297.

<sup>6</sup> *Lancet*, 1909, i, p. 1105.

<sup>7</sup> *Journ. Amer. Med. Assoc.*, 1912, lix, p. 718.

A very interesting point in this last case was that the family history disclosed the fact that four other members of the family seemed to have suffered similarly with hæmatoporphyrinuria and nausea and abdominal pains, one of her sisters having died after being ill for nine months with similar symptoms. The strong family tendency to these symptoms with hæmatoporphyrinuria in these cases is practically conclusive evidence of an autotoxæmia being the origin of the syndrome. In all probability an intestinal toxin is to blame, and Barker and Estes regard the gastro-duodenal dilatation as primary, the toxæmia producing the hæmatoporphyrinuria and polyneuritis being secondary. In my second and fatal case the obstinate constipation was a striking and troublesome symptom, from which he had always suffered, and it is suggestive that an enterotoxin was here responsible for the acute illness, though what was the actual exciting factor in precipitating the attack is a mystery.

Günther applied the term "acute toxic hæmatoporphyrinuria" to those cases in which the symptoms follow chronic poisoning by sulphonal, trional, tetronal, veronal or its derivatives. Other substances such as lead, saffron, and glycerine are also said to produce toxic hæmatoporphyrinuria.

In 1896, while house physician at the National Hospital, I saw hæmatoporphyrinuria followed by increasing weakness, flaccid paralysis, and death in a woman who had been taking 20 gr. of sulphonal daily for three months. Recently I have had a case of slight hæmatoporphyrinuria in a man aged 65, with increasing pains in the legs, who has been taking from 5 to 7 gr. of medinal nightly for five months.

Many cases of this acute sequel of chronic sulphonal poisoning have been published since that of Stokvis in 1889, and in about one-fourth of the cases acute paralysis has been noted. The syndrome closely resembles that of acute hæmatoporphyrinuria not due to drugs, as already described. Thus, severe abdominal pains, vomiting and constipation are often prominent symptoms, recovery rarely taking place when all these three symptoms are present.

A typical case was described by Anson,<sup>1</sup> which he saw in 1901, that of a woman, aged 35, who after taking 30 gr. of sulphonal daily for three and a half years, developed acute abdominal symptoms, constipation, hæmatoporphyrinuria, intense pains in the legs, loss of knee-jerks, followed ten days later by marked weakness of the legs, increasing to total paralysis, the arms being soon similarly affected. There was no anæsthesia, but incontinence of both sphincters until death.

In Keith Campbell's case, published in 1898, the paralysis first appeared in the arms, before the legs were affected, and convulsions and nystagmus were noted, death occurring thirteen days after vomiting commenced.

In most of the toxic, or sulphonal cases, there was paralysis: this occurred first in the legs, while in the acute neuritis accompanying acute hæmatoporphyrinuria not due to drugs, including my own two cases, the neuritis has appeared usually first in the arms. Such development of the weakness, especially when followed by atrophy, excludes Landry's paralysis, while the symmetry of the paralysis and the absence of the deep reflexes indicate polyneuritis, not poliomyelitis. It is remarkable how often involvement of the sphincters has been met with in hæmatoporphyrinuric polyneuritis: it was present in my own two cases.

<sup>1</sup> *Brit. Med. Journ.*, 1920, i, p. 634.



## DIFFERENTIAL DIAGNOSIS.

The differential diagnosis of polyneuritis from acute poliomyelitis and from Landry's paralysis is sometimes extremely difficult. The separation from poliomyelitis in some cases is all the more difficult because of the combination of the two conditions that occasionally occurs. Paræsthesiæ and peripheral anæsthesia will probably indicate the presence of polyneuritis in a doubtful case, while an extensor plantar reflex will be evidence of cord involvement. Asymmetry of the paralysis certainly does not exclude polyneuritis, wasting of the intrinsic musculature of the hands is also common in polyneuritis, and retention of the knee-jerks is by no means rare. Sphincter paralysis is rare in both poliomyelitis and polyneuritis, but is occasionally met with in both these diseases.

Diffuse sarcomatosis of the cerebro-spinal meninges may at first simulate polyneuritis.

The differences in extent of involvement of the sensory nerves on the one hand and of the motor nerve fibres on the other in polyneuritis is remarkable, though not constant for the same toxic cause. Lead is almost purely a motor nerve poison, but diabetes may cause extremely painful local neuritis, or generalized sensory symptoms and ataxy, without any pain. No sensory symptoms whatever occurred in my case of carbon bisulphide polyneuritis. Rheumatic forms of neuritis are usually very painful, so are the alcoholic, so also are tuberculous neuritis of the feet, and the neuritis of malignant disease. Hæmatoporphyritic neuritis is usually very painful, though other forms of autotoxæmic polyneuritis may show little or no sensory involvement, and may thus be mistaken for poliomyelitis or Landry's disease.

Numerous post-mortem accounts have been published of polyneuritis in which there has been found more or less profound degeneration of anterior horn cells, and indeed it is not surprising that, in a severe case of polyneuritis with considerable muscular atrophy lasting for many months, changes should be found in the anterior horn cells.

Twenty years ago Stanley Barnes published in *Brain*<sup>1</sup> such cases under the title of "Toxic Degeneration of the Lower Neurones," and sought to distinguish them from polyneuritis by (1) the great atrophy of the intrinsic hand muscles; (2) the comparative slowness of the sensory changes; (3) the absence, as a rule, of contractures, and (4) the integrity of the psychical condition. All these points seem to be comparatively common in cases which clinically should undoubtedly be classed as polyneuritis. A not irrational view perhaps is that which regards polyneuritis and poliomyelitis as essentially the same process affecting different portions of the same neuron; and there can be no doubt that occasionally mixed cases are met with, of permanent cord damage associated with polyneuritic symptoms, which recover.

<sup>1</sup> *Brain*, 1902, xxv, p. 479-500.



## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### Case of Tremor for Diagnosis.

By ANTHONY FEILING, M.D.

PATIENT, a girl, G. C., aged 10½, was brought to hospital on account of involuntary movements of the left side of the body. She was a full-time child, a breech presentation, and at birth had white asphyxia. Sat at the age of 18 months, walked at 3 years. Has never had fits. No illnesses except measles. At age of 9 months involuntary movements were noticed on the left side of the body, with weakness in holding up the head. During the last 5 months these movements have become worse and have extended to the other side of the body. At times her voice becomes very weak. No history of any nervous disease in the family. The child is well grown and appears quite intelligent.

There is a slight weakness of the lower part of the right side of the face, and the tongue is deviated slightly to the left. The left arm is the seat of a rhythmical tremor, more marked in the proximal part of the limb. The movements produce rotation of the arm at the shoulder, pronation and supination of the forearm at the elbow, and flexion and extension of the fingers. The rate is about two to the second. Both arms are hypotonic. Similar movements affect the trunk and the legs, though to a lesser extent. In the legs again the proximal muscles are involved to a greater extent than the distal. There is no actual paralysis, though the power of the left arm and leg are less than on the right side. The deep reflexes are present; the plantar responses are flexor.

The Wassermann reaction in the blood is negative, and the cerebro-spinal fluid shows no abnormality.

### Case of Unusual Sequelæ of Lethargic Encephalitis (Parkinsonian Syndrome associated with Right Hemiplegia, showing Peculiar Disturbances of Tone and Posture in the Limbs on the Hemiplegic Side).

By DOUGLAS McALPINE, M.B.

PATIENT, a female, A. F. C., aged 20, was quite well until April 18, 1919, when she developed a typical attack of encephalitis lethargica, complicated from the outset by a right hemiplegia. Her present condition gradually developed within a few months from the onset.

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The following is a brief summary of her condition: Parkinsonian facies, smile and speech; slight right hemiplegia with definite extensor response on right side; on left side doubtful extensor response. Power quite good in all four limbs; no sensory disturbances.

Tone: At proximal joints in all four limbs, slight resistance to passive movements both of flexion and extension, most marked in the lower limbs. If fingers or wrist of the right hand be passively moved into any position, such position is maintained involuntarily. The right ankle when inverted or everted remains in that position; this is not seen however in the dorsi flexors or plantar flexors, nor in the limbs on the left side.

Voluntary movements: Extension at the elbow is only carried out in the normal way when the fingers are extended: with fingers flexed there is always accompanying pronation: extension of the fingers is only possible either by the patient voluntarily abducting the arm, flexing the elbow, pronating (moderately) the forearm and flexing the wrist, or by extension at the elbow with hyperpronation.

The posture of the hand and wrist on voluntary movements tends to take one of four patterns: (1) Flexion of fingers and wrist. (2) Extension of fingers and thumb: wrist either flexed or slightly extended. (3) Extension of wrist and first phalanges, with flexion at interphalangeal joints and apposition of thumb. (4) Same position in fingers as in (2), but with flexion of wrist and pronation of forearm. The position of the elbow is variable.

All voluntary movements with the right hand are carried out slowly and with difficulty. The phenomenon of tonic innervation is well seen in the right hand and wrist. If the patient is asked to grasp the observer's hand, and is then told to relax her grip she is unable to do so for a period varying between fifteen seconds and two minutes: she has to manœuvre her arm into the position described above as necessary when extending the hand. This condition is not seen at the proximal joints, but is present to a slight extent at the right ankle. There are no athetotic movements nor motor apraxia.

A flexion, but sometimes extension reflex (as described by Riddoch and Buzzard [1]) is obtainable in both upper limbs as a result of a nocuous stimulus applied to either the extensor or the flexor aspect of the arm.

On assuming the upright posture the right arm at once becomes extended at the elbow, hyperpronated, flexed at the wrist, which becomes deviated to the ulnar side: the fingers are flexed into the palm. As soon as the right foot touches the ground it becomes powerfully inverted with plantar flexion of foot and toe, the hallux being dorsiflexed, so that the pad of the toes alone touches the ground; at the same time there is marked extension at the knee; after half a minute or so the tonic spasm of the foot gradually relaxes and the right foot assumes its correct posture on the ground. On attempting to walk the right leg is over-flexed at the hip and the whole leg goes into extensor spasm again as above described; the attitude of the leg at this stage resembles that depicted in the goose step of the German soldier. Gradually the right leg is lowered and the toes approach the ground and then, as the spasm relaxes, the heel is gradually lowered until it touches the ground, the weight meanwhile being transferred to the right leg. As the right leg is carried forward, the right arm, which maintains the posture described above with palm turned outward, is powerfully retracted and abducted at the shoulder, and as the patient transfers her weight on to the right leg the right arm comes forward again into line with the body.

## REMARKS.

In this case there is evidence of a moderately well developed condition of Parkinsonism associated with slight right hemiplegia, and in all probability some disturbance of the left pyramidal tract, as evidenced by the nature of the plantar response with a degree of flexion, withdrawal of the limb, and, in addition, the presence of a well marked flexion reflex in the left upper limb.

Despite this bilateral pyramidal involvement there is an entire absence of pseudo-bulbar symptoms. There is no evidence in this case that the phenomenon of tonic innervation depends on a lesion on a higher plane than the internal capsule; this, however, was the case in seven out of the nine cases collected by Wilson and Walshe [2]. The condition seems to indicate lack of reciprocal inhibition of antagonists, so that innervation in one muscle group persists in spite of volitional attempts to innervate the opposing group of muscles as was suggested by these authors. They laid considerable stress on the exclusion from the term "tonic innervation," of conditions resulting from involuntary or reflex movements. In this case, apparently, the same phenomenon is present both on voluntary and passive movement; so that no separation is possible between the phenomena occurring on voluntary and involuntary movement, and it seems certain that in this case the condition of tonic innervation can be explained by a central lesion resulting in a disturbance of reflex tonus. The posture on the right side, which results when standing or attempting to walk, is clearly determined by the assumption of the upright position and resembles closely the antigravity posture as described by Sherrington as present in the decerebrate animal [3].

I am indebted to Dr. H. Campbell Thomson for permission to show this case.

## REFERENCES.

[1] RIDDOCH, G., and BUZZARD, E. FARQUHAR, *Brain*, 1921, xliv, pp. 397-489. [2] WILSON and WALSH, *Brain*, 1914, xxxvii, pp. 199-246. [3] SHERRINGTON, C. S., *Journ. Physiol.*, 1897-98, xxii, pp. 319, 320.

## DISCUSSION.

Dr. WILFRED HARRIS (President) asked if the case was progressive so far as the Parkinsonian movements were concerned.

Dr. MCALPINE replied that there was no great interval in his case between the attack of encephalitis and the onset of the present disorders of movement. He did not think that all the symptoms in this case could be accounted for by the superposition of hemiplegia on the Parkinsonian syndrome.

## Complete Amaurosis, Dementia and Spastic Paralysis in a Hebrew Boy, aged 10.

By GEORGE RIDDOCH, M.D.

FAMILY history: Father and mother first cousins. No history of nervous troubles in relatives on either side of the family. Three children: (1) Boy, aged 12, normal; (2) patient; (3) girl, aged 7, normal.

Previous history: Birth at full term. Normal labour. Breast fed until 18 months old. Began to walk at 9 months. Could say "mammy" and

### 30 Riddoch: *Complete Amaurosis*; Collier: *Sclerodermia*

"daddy" at 12 months. Measles in second year. Apparently bright and intelligent child until the age of 4 years.

Present illness: At the age of 4 years progressive weakness of lower limbs. At 6 years weakness of upper limbs, and defective speech first noticed. Mental deterioration, destructiveness and screaming fits. After an attack of influenza disabilities rapidly increased. At the age of 8 years, it became evident that he could not see well. Completely helpless; incontinent; had to be fed. Fits are frequent, and there is obstinate constipation.

Present condition: Complete dementia, blindness, and spastic paralysis; upper limbs in flexion and lower limbs in extension. Lies on his back with head to one side and saliva dribbling from his open mouth. Quick involuntary flexor movements, especially of upper limbs, occur frequently. These myoclonic movements can be evoked by the slightest tap on any part of the body, and sometimes by a loud noise. They are often bilateral and symmetrical, and are then accompanied by movements of the head and face, contraction of the muscles of the trunk and a grunting noise. All tendon reflexes are much increased, and the plantar response is extensor on both sides. Bilateral optic atrophy with much pigmentation of the retina. No cherry-red spot in the position of the macula is seen on either side. The Wassermann reaction of the blood was reported as being doubtfully positive.

#### DISCUSSION.

Dr. WILFRED HARRIS (President) remarked that the retinal changes resembled retinitis pigmentosa, and it was to be noted that the parents of the child were first cousins.

Dr. GORDON HOLMES considered that there was some doubt as to the nature of the disease in this child. The type of idiocy and blindness described by Spielmeier resembled amaurotic family idiocy in many respects, whereas the Batten-Mayou type was different. In this case the appearance of the fundus was different from that seen in the Batten-Mayou type, and resembled more closely the condition in Spielmeier's type, which was supposed to be *retinitis pigmentosa sine pigmento*. The retinal changes in this patient strongly suggested syphilis, either of congenital origin or acquired early in life. On the other hand the long duration and slow progress were against a syphilitic cause.

Dr. F. PARKES WEBER suggested that the case might (in spite of the Wassermann result) be one of juvenile general paralysis, with an optic nerve atrophy as in tabetic cases—in fact, a "juvenile tabo-paresis."

Dr. FEILING said that he had seen, in a case of tabes, a similar fundus oculi which closely resembled the condition in retinitis pigmentosa.

Dr. RIDDOCH replied that there was no definite evidence of syphilis in this case. There had been no miscarriages, and the other children were perfectly normal.

#### Case of Sclerodermia.

By JAMES COLLIER, M.D.

(Shown by J. P. MARTIN, M.D.)

PATIENT, a female, M. C., aged 43. In March, 1921, patient had an illness which was stated to be influenza. In May she noticed she had difficulty in kneeling on account of pains in the thighs and back. In November she began

to feel stiffness of the legs after standing a little while, and this has continued up to the present. She complains of no pain except in thighs and back after standing or walking. Her periods have been irregular since January, 1921.

Present state: Rather emaciated. Since her illness in March, 1921, her weight has fallen from 8 st. odd to 6 st. odd. Hair lustreless and coming out considerably. High complexion. Skin of nose shiny and red. Lips puckered. There is some weakness of the lips shown in whistling and when the cheeks are blown out. Below the chin and on the anterior surface of the neck no subcutaneous tissue is palpable. Skin and subcutaneous tissue of the cheeks feel tough. Scarcity of fat padding all over the body. Marked osteo-arthritis most noticeable in the hands, knees and feet. They are all quite painless, and patient was unaware of them until her attention was called to them. The fingers, are short, podgy and swollen, cyanosed at the proximal phalangeal joints. The skin over them is shiny and hard and difficult to pick up. The little fingers have flexor contractures. The feet and ankles are slightly swollen, the feet tend to be flat, and they sweat a good deal, especially the left. The skin over the toes and anterior part of the feet feels hard, and the toes are cyanosed. Circulatory system: frequent extrasystoles. The nervous, respiratory and alimentary systems are normal.

### Case of Sclerodermia.

By T. GRAINGER STEWART, M.D.

(Shown by J. P. MARTIN, M.D.)

PATIENT, a male, H. W., aged 46, in September, 1921, felt a pain in the middle of the left shin. About a month later he began to experience a feeling of "pins and needles" in the right hand affecting all the fingers, so that writing was uncomfortable; he could feel it creeping up his hand day by day. After a very short time the left hand became affected and then the right shin became painful just as the left.

Practically from the beginning, swelling occurred in the hands and feet. The swelling of the hands was not great but was fairly constant and chiefly affected the fingers; it was worse when the hands became cold. The swelling of the feet came on in the evenings after patient had been about all day; it was much greater in amount than the hand swelling and it affected the whole of each foot and ankle; it had always completely disappeared in the morning.

About Christmas, 1921, he suffered for a month or so from pains throughout all four limbs; they came on at night as soon as he got warmed up in bed.

For two or three years he has had a small firm swelling as large as a bean on the inner border of the left forearm. More recently other similar swellings have come up on both forearms and one on the right thigh. There are none on the left lower limb.

For the past year he has not been able to close his hands so well as before; this limitation of movement has gradually become worse, so that now he can only flex the fingers to the extent of touching the intrinsic joint of the thumb.

Present state: In the subcutaneous tissue of both forearms in the line of the median and ulnar nerves and also in the outer border of the left forearm there are a number of small, firm swellings, varying from the size of a pea to rather larger than a bean. They are attached to the skin and to the surrounding tissue, and feel fibrous. The skin over them is not reddened and there is no

appearance of any inflammatory reaction. Similar nodules are present, one in the left iliac fossa, one in the right hypochondrium, one on the anterior surface of the right thigh. On the forearms and legs the skin is hard, tight and firmly bound down to the surrounding tissues; only with difficulty can it be pinched. On the legs it is glazed and marked by numerous scratchings. The tibiae are tender, especially along the anterior borders; there is some tenderness also of the femora. Muscles: The muscles of the forearm, even when not in contraction, feel hard, the hand-grips are strong in gripping large objects, very feeble when gripping small ones because of the limitation of movement of the fingers. There is slight hardening of the muscles of the upper arms. In the lower limbs all movements are strong, but there is limitation of movement of the left ankle due to the stiffness and want of elasticity of the muscles and subcutaneous tissues. The calf muscles even when relaxed are very firm and the skin is drawn tight over them, there is no wasting or fibrillation. There are no changes in the reflexes, and no sensory loss. The cerebro-spinal fluid is normal, and the Wassermann reaction negative in blood and cerebro-spinal fluid. There are no X-ray changes in the bones or periosteum. The electrical reactions of the muscles are normal.

One of the nodules has been dissected out from the left forearm. During the dissection a dense fibrous aponeurosis was seen tightly ensheathing the muscles. The muscles below this appeared normal.

#### PATHOLOGICAL REPORT.

The rounded mass consists of a nodule of fat contained in a capsule of dense fibrous tissue which is infiltrated with small rounded cells and fibroblasts.

#### DISCUSSION.

Dr. PARKES WEBER said that these cases must not be confused with acute or subacute polymyositis, or poly-dermatomyositis, which was an acute infectious disease in which there was fever and which had sometimes been confused with trichinosis in countries where trichinosis occurred. In the present case the muscle seemed little affected, and the condition appeared to be allied to the generalized symmetrical form of sclerodermia. He noted that there was evidence of improvement and would like to know what treatment had been employed.

Dr. WILFRED HARRIS (President) described a case of sclerodermia which started with a sudden attack of vertigo. Later the sclerodermia disappeared and left the patient with Erb's juvenile type of myopathy.

Dr. J. P. MARTIN replied that the treatment in Dr. Collier's case had been thyroid gland by the mouth, massage, and sinusoidal baths to the hands and feet.

### Tumour in Cisterna Magna.

By J. P. MARTIN, M.D., and J. G. GREENFIELD, M.D.

(Case of Dr. JAMES TAYLOR, C.B.E.)

HISTORY: Patient at time of admission complained of paralysis of both arms and both legs, and had been unable to walk for three months. Three years ago patient suffered from "pins and needles" in third, fourth and fifth



fingers of left hand; this feeling was not present constantly, and most often occurred in the morning. The "pins and needles" spread slowly over the back of the left hand and up the left arm, and he became unable to grasp things with the left hand. In July, 1920, he went to St. Thomas's Hospital and was treated as an out-patient for six months. During that time the left arm got no better and the right began to be affected with the same feeling, but in it the feeling spread from above downwards.

On January 20, 1921, he was admitted to St. Thomas's; before that his legs had been a little shaky but were still strong and did not cause any disability in walking. On admission he was put to bed and four days later when they got him out of bed to weigh him he "could not find any legs" and was unable to stand at all; he could still use his arms and feed himself.

After three months' treatment he was able to walk again, half a mile or so with assistance, and he was discharged on April 8, 1921. He became short of breath about July, and when attending as an outpatient in August, 1921, he had an attack of breathlessness and was readmitted. Whilst in hospital this second time his breathlessness became less, but by remaining in bed he gradually got weaker and became unable to walk except for a few steps. He was sent home in an ambulance in October, 1921.

For the last six months he had been at home and had become rather worse; he had not walked at all for three months. He had been accustomed to get up every day and sit in a chair from 9 o'clock in the morning till 9 at night, being helped out of bed and put back by two friends.

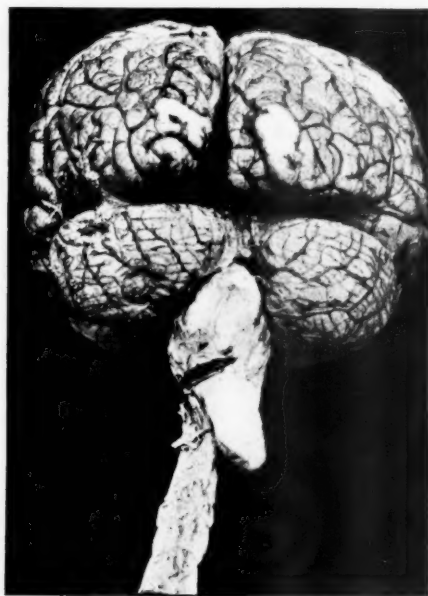
For nearly eight months (since middle of last stay in St. Thomas's) he had been unable to feed himself or use his arms in any way. No trouble with water. Insufficient control of bowels. He had never suffered any pain in body till last few weeks; on admission he had occasional pain in back of neck. On about a dozen occasions during last three months he had had double vision.

State on admission to the National Hospital: Patient was a man, aged 45. Lips and face cyanosed. He was very helpless and quite unable to move himself in bed. He could not raise his head from the pillow. He had a frequent cough. There were occasional râles and rhonchi over the chest. He was unable to expand his chest owing to paralysis of trunk muscles. Pulse-rate usually about 96 per minute. The urine contained no albumin. Psychic functions: Intelligence clear; patient gave a good account of his illness. He was querulous and difficult to manage in the ward. No aphasia. No articulatory defect. Cranial nerves: Discs healthy. Pupils equal, regular, react normally. Ptosis both sides, more right than left. External ocular movements full. No diplopia on examination. A few nystagmoid jerks in both eyes on extreme deviation to either side, but especially to the right. Sensation over face normal. Jaw movements strong. Corneal reflex present right and left. In showing teeth left side of mouth not so well retracted as right. Forehead wrinkled and eyebrows raised to compensate for ptosis. Weakness in turning head to either side, especially to left. Backward movement weak also. Sensory functions: no loss to pinprick, cotton wool, heat or cold. Vibration sense lost in both arms and both legs. Sense of position lost in the fingers and in the large joints of right arm; not completely lost in left arm; lost in the toes of both feet and in the ankles. Complete astereognosis. Motor system: power very variable; after patient had been sitting up for a time he was at his best, and became very helpless after lying in bed. When patient sat in a chair his head fell forward, he could raise it, but soon got



34 Martin and Greenfield: *Tumour in Cisterna Magna*

tired holding it up. He could only sit in a chair with support, he could not sit up in bed. Muscles of abdomen, chest and back, flabby. There was possibly slight wasting above clavicles. No fibrillation. Both upper limbs were powerless for practical purposes. Arms lay slightly flexed at elbows and wrist, fingers half flexed. Considerable power of lower limbs was present after he had been sitting up. Movements of the left limb generally weaker than those of the right. Extensor movements strong at knee and ankle, flexion movement weak; power of movement of the toes was fair. The hands were cold and blue and the fingers swollen. Reflexes: Arm-jerks all brisk. Knee-jerks + + +. Ankle-jerks present with clonus both sides. Sup. abdominals absent. Plantars extensor right and left. Organic: no incontinence or



Tumour in cisterna magna.

retention but defective control over both vesical and anal sphincters. Wassermann reaction negative. No change in electrical reactions of muscles. Nothing abnormal in radiogram of cervical spine.

The patient died of hypostatic pneumonia. At the post-mortem examination a large tumour was found filling the cisterna magna.

DESCRIPTION OF SPECIMEN.

The tumour, which weighs 32.5 gm., is firm in consistence and in shape like a pear. It fills and distends the cisterna magna, lying altogether between the arachnoid and the pia mater. The larger end shows a small knob on its

upper and posterior surface where it presses against the right lobe of the cerebellum. The tumour lies against the foramen of Magendie, but has caused little hydrocephalus as the foramina of Luschka are unusually wide. The posterior surface of the medulla, from the tip of the calamus scriptorius downwards, is compressed forwards, so that it appears hollowed out. This pressure affects particularly the cuneate and gracile nuclei, the restiform bodies being only compressed at their posterior border. At the lower end of the medulla the tumour passes to the right of the mid-line so that the right dorso-lateral surface of the first cervical segment is somewhat compressed. The cord at this level is much softened.

Microscopical sections show the tumour to be a typical fibrous meningeal endothelioma.

## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### Dystrophia Myotonica (Myotonia Atrophica), an Heredo-familial Disease with Cataract.

By W. J. ADIE, M.B.

IT happened about a year ago in the course of routine out-patient work that my first patient on two consecutive days suffered from a disease which I was then content to call myotonia atrophica; and when I related this coincidence to a colleague at another hospital a day or two later I was promptly invited to "come into the wards and see two more."

Naturally, the lively interest thus created by chance supplied the stimulus for further investigation. I can remember seeing one case only before the incident I have related, but since that time I have come across twelve more, many of them in the course of routine work at general hospitals. In all but four cases, which I saw by the courtesy of colleagues, the trouble was either unrecognized, the patient being under treatment for some unrelated condition—on the surgical side in one instance—or it was labelled progressive muscular atrophy or myopathy or, in one case, myasthenia gravis. I was not surprised then, in my search for further information, to find that the number of cases recorded already runs into hundreds, and that much more is known of this interesting and many-sided disease than is indicated by the meagre descriptions that exist in the English language. So far as I know, the subject has not been discussed at length by this Section for many years, and although the foreign literature is extensive, little has been written upon it in this country since the late Dr. Batten made it known to us some thirteen years ago. This would not be surprising if dystrophia myotonica were a very rare disease or one of which little remained to be said; but it is certainly far from rare in comparison with many nervous and general diseases the characters of which are well known to us; and I trust that what I have to say will convince you that much remains to be said before the problems are solved that it presents not only to neurologists but also to ophthalmologists, to physicians who interest themselves in disorders of internal secretion, and to those who make a study of heredo-familial disease.

The *history* of dystrophia myotonica may be said to begin in 1886 with the appearance of Erb's work on Thomsen's disease, for although the two conditions are quite distinct, Erb's work, by arousing widespread interest in myotonia, led to the publication of descriptions of a large number of cases of "atypical Thomsen's disease"—so called because myotonia was a symptom common to them all—and it was from this heterogeneous mass that the group of cases which now concerns us finally emerged.

[December 14, 1922.]

The first clear descriptions of the distinctive features of this group were given independently in 1909 by Batten [1] in England and by Steinert [2] in Germany. These observers agreed on many points—on the peculiar distribution of the muscular atrophy in particular—but whilst Steinert maintained that myotonia was the initial and essential symptom and that atrophy was merely an incident in the course of Thomsen's disease, Batten held that myotonia was "merely a symptom," and he placed his cases in close relation to the myopathies.

Time has confirmed the accuracy of their description so far as the muscular symptoms are concerned, but it has also led to a complete change in our conception of the nature of the disease, which, far from being one of muscles alone, must now be regarded as a general disease with widespread manifestations.

This change was hastened by the description given by Greenfield [3] in 1911 of a family of thirteen brothers and sisters, in which two suffered from myotonia atrophica with cataract, three from myotonia atrophica alone, and two from cataract alone. Later in the same year Ormond [4] in this country, and Kennedy and Oberndorf [5] in America, described cases of myotonia atrophica with cataract, and after that the combination was frequently encountered. At once the *other* extra-muscular symptoms, atrophy of the testicles, loss of hair and the rest, which had been noticed before but had been regarded as fortuitous, were viewed in a new light; in 1912 Curschmann [6] raised them to a cardinal position and insisted that they must receive full consideration in any attempt to explain the disease. Since then closer study has added considerably to our knowledge and has led Naegeli [7] to assign both myotonia and muscular atrophy to a position subordinate to the extra-muscular symptoms. This standpoint—that dystrophia myotonica is essentially a pluriglandular syndrome, a disease of internal secreting glands—is one that we shall have to consider later.

These changes of view are reflected in the names that have been applied to these cases. The original designation "Thomsen's disease with muscular atrophy" is a reminder of a time when any patient with myotonia, but of a type deviating in any respect from Erb's classical description, was described as an atypical case of Thomsen's disease. In 1901 the name myotonia atrophica was proposed by Rossolimo [8] for those cases in which atrophy was supposed to have supervened in Thomsen's disease, and this name has been retained in this country, although the separation between the two diseases was made more than ten years ago. From every standpoint the name is unsuitable. It suggests that myotonia is *the* constant and essential symptom, that myotonia precedes atrophy, that myotonia and atrophy appear in the same muscles, that the disease has some connexion with Thomsen's disease, and that it is a disease of muscles alone. But myotonia is not constant, it is often preceded by atrophy, it is usually found in muscles free from wasting, myotonia atrophica has nothing to do with myotonia congenita, and extra-muscular symptoms are always present. For these reasons I prefer to use the name proposed by Curschmann, especially because it emphasizes the dystrophic nature of the disease.

Unfortunately, finality in nomenclature has not been reached, for, as you will agree later, we still require a name which will designate the heredo-familial *disease* of which the *syndrome* which now concerns us is merely one aspect.

The name used by Naegeli, "the pluriglandular disease of internal

secreting organs" is not justified at present, but we shall find ourselves obliged to consider the ideas it connotes when we come to discuss pathogenesis.

*The Symptoms.*—Here a recapitulation and, apart from cataract, a brief discussion of some of them is all that is required, as most of you are already familiar with them. The onset is most frequent between the ages of 20 and 30, males being attacked more often in a proportion of about five to one. The symptoms fall roughly into four groups:—

(1) The muscular symptoms: muscular atrophy and myotonia.

(2) General dystrophic symptoms: a useful but somewhat loose designation for a host of phenomena suggestive of disturbances of internal secretion or of sympathetic innervation. It includes cataract, atrophy of the testicles and loss of sexual power, loss of hair, especially frontal baldness, general loss of body weight much greater than can be accounted for by loss of muscle substance, changes in the skin, bones, nails and teeth, increased secretion of sweat or tears or sputum, Chvostek's sign, vasomotor disturbances such as cyanosis and coldness of the extremities, abnormal reactions to injections of atropine, adrenalin and pilocarpine, and many other symptoms which are supposed by enthusiasts to indicate vagotonia or sympatheticoetonia.

(3) The so-called tabetic symptoms: loss of tendon reflexes, ataxy, pains, and sensory disturbances.

(4) Other symptoms: the peculiar and almost constant speech defect, psychical defects, stigmata of degeneration, especially a narrow high-arched palate, and other occasional symptoms too numerous to mention.

Of *muscular atrophy* I need say but little. Its distribution is peculiar to this disease from the fact that it always appears first in one of the following groups of muscles: (1) The head and neck group—that is, the muscles of the face, the muscles of mastication, and the sternomastoids and deep neck muscles; or (2) the muscles of the forearms; or (3) the muscles on the anterior and lateral aspects of the leg. It is usually found in more than one of these groups when the patient is first seen, and the order just given—head and neck, forearms, legs—is that in which it is most frequent.

In the head and neck group it is usual to find some weakness in all the muscles mentioned, but one often suffers more than another, and atrophy may be extreme in one before the others are attacked. I should say that absence of some weakness in closing the lids, that is absence of some indication of the facies myopathica, is the rarest negative finding, absence of atrophy or weakness of the sternomastoids being next in order of rarity. The temporals often show extreme wasting when the masseters retain their bulk and power, and the deep temporal hollow so produced aids materially in producing the characteristic facies of the disease. In the forearms the supinator longus nearly always suffers first and most. It is said that the extensors always waste earlier and more than the flexors, but I have seen several cases in which the reverse was true. In the leg the anterior tibials and peronei almost always succumb first. From these points of predilection atrophy may spread to any other muscle, and it may be found outside the nucleus before all the classical groups are attacked. For example, when atrophy in the forearms is severe the small muscles of the hand may waste before the legs are attacked, but atrophy never appears *first* in any muscle outside the classical groups. In some advanced cases every muscle in the body is wasted, in others the trunk muscles and the muscles of the shoulder and pelvic girdles retain their bulk. In some cases, few in number but of great theoretical interest, atrophy and weakness were entirely absent.

In these the diagnosis was suggested by the presence of a number of other characteristic symptoms and was confirmed by the occurrence of the fully developed disease in other members of the family.

*Myotonia* is shown by delayed relaxation of muscular contractions produced voluntarily or by mechanical or electrical stimulation. It may be present in any one of these forms alone. According to the classical descriptions active myotonia is almost always confined to the handgrasps, but my own observations have convinced me that it is often more widespread. It is true, however, that in this disease, in contrast to Thomsen's disease, it is either confined to or most conspicuous in the hand-grasp in a very high majority of the cases.

Mechanical myotonia, shown by delayed relaxation of contractions produced by striking the muscle with a percussion hammer, is often of much wider distribution. It is most constant in the tongue and in the small muscles of the hand, and is sometimes confined to them, but in many cases it can be demonstrated in most of the limb muscles in which atrophy is slight or absent. This does not mean, as some have stated, that there is any antagonism between atrophy and myotonia, that myotonia does not occur in those groups which usually waste, for I have seen prolonged myotonic contractions in these muscles in cases in which atrophy happened to be slight or absent. The characteristic reaction is the formation of a dimple on the lateral border of the tongue, or furrow in the length of the fibres of larger muscles; this must be distinguished from the persistent elevation at the point of contact which can often be evoked by percussion of normal muscles.

Myotonia is usually difficult to demonstrate on electrical stimulation and its distribution is always sparse. The electrical reactions show many variations from the classical myotonic reaction of Thomsen's disease. I cannot discuss them here.

I have found myotonia a most fascinating symptom and I dismiss it thus summarily with some regret. This much must be said—in a few cases it has been absent on repeated examination where other symptoms and the family history made the diagnosis certain.

Of the extra-muscular symptoms *cataract* must be given first place, not because it is most frequent (other symptoms in this group appear more often), but because of its great interest. In its typical form it begins as a posterior lamellar cataract with star-shaped processes. As a rule the anterior lamellæ next become opaque, and in most cases, with or without cortical changes, numerous very fine opacities are found scattered throughout the lens. It ripens quickly to a total soft cataract with a soft nucleus, at about the same time in both eyes. It is not possible at present to state with certainty the proportion of cases in which it occurs, because it may be symptomless in the early stages (I saw a good example of this a few days ago) and a special examination of the lens has only recently become a part of the routine examination of these cases. Curschmann's [9] estimate is 30 per cent., I am inclined to think that it will turn out to be higher than this.

But the importance of cataract in this disease lies not so much in its coincidence with the other symptoms, though this is remarkable in itself, but in the fact that it often occurs in otherwise healthy members of the dystrophic generation, and more remarkable still, it is common in otherwise healthy members of preceding generations. In Greenfield's now famous family two otherwise healthy members of the dystrophic generation—that is the generation in which dystrophia myotonica occurred—had cataract, likewise a paternal

aunt and grandmother. In one of Curschmann's families cataract was found in one otherwise healthy member of the dystrophic generation, in four members of the preceding generation and in one of the generation before this. The same phenomenon has been reported by Ormond, Hoffmann, Hauptmann, Rohrer, Higier and others, but instead of multiplying isolated instances I shall draw your attention to the observations of Fleischer [10] who has met with no less than sixty-eight cases of dystrophia myotonica, including thirty-eight with cataract. This large material was discovered in the main by appropriate examination of patients who had come to the eye hospital in Tübingen, a town of some 30,000 inhabitants, for treatment of presenile cataract. May I say, in passing, that I find in this experience justification for a plea that our ophthalmological colleagues will send their patients with presenile cataract to a neurologist with a view to an examination for signs of dystrophia myotonica and an inquiry into the condition of other members of their families.

Fleischer's thirty-eight cases with cataract were distributed over twenty-seven families. In eleven instances a parent had cataract, it was common in aunts and uncles, and also in still earlier generations. In one family in which three brothers and sisters transmitted the disease to their offspring these three, their younger brother, a sister of the grandfather and a daughter of his sister as well as the common ancestor—the great grandfather of the dystrophic generation—all had cataract.

Another noteworthy point is the age at which the cataract appeared, or at which an operation for it was performed. In Curschmann's family the ages are 38, 42, 44 in the dystrophic generation, and 50 in the parents, with senile cataract in the earlier generation. This "anticipation," which I understand also occurs in other forms of hereditary cataract, was seen in eleven of Fleischer's families, and is well illustrated in one in which the ages are 27, 30, 31 and 40 in the dystrophic generation, and 37, 38, 52, 56 and 65 in the preceding generation, with ordinary senile cataract in the earlier generations.

These examples will, no doubt, suffice to convince you that cataract is a feature of this disease, and that the heading I chose for my paper—an heredo-familial disease with cataract—has something to justify it.

The disease is familial and hereditary in every instance, but isolated cases seem to occur because it often happens that in large families few members are affected. The incidence in certain families has consisted in one child in a family of nine, two in fourteen, two in twelve, two in eleven. In one family only one child of eight examined had dystrophia myotonica, but the familial nature of this case was proved when it was discovered that this patient and another apparently isolated case had a common great-grandfather. In two of Fleischer's families the relationship of apparently unconnected families went back to a common great-great-grandfather and in two more to a common great-great-great-grandfather, the intervening generations being healthy so far as is known, apart from the occasional occurrence of cataract.

We are faced then with the highly remarkable fact that this disease may be transmitted through as many as six generations, to burst forth suddenly in a number of families at the same distance from a common ancestor. I have made a careful study of all the case reports available, for acceptable evidence of muscular atrophy or of myotonia in generations preceding or succeeding the dystrophic generations, and, except for one observation by Grund, have found none whatsoever. It seems then that the syndrome we call dystrophia myotonica is usually confined to one generation, although abundant evidence—the occurrence of cataract in preceding generations and other facts that I have



not mentioned—proves that some branches of the family are undergoing steady degeneration. Time alone will enable one to tell whether the disease does indeed die out, as it seems to do, with the dystrophic generation. All I know is that records exist of about one hundred children born to parents who developed the dystrophic syndrome, and a number of grandchildren, and that none of them has suffered from muscular atrophy or myotonia or cataract. The son of one of my patients, a professional long-distance walker of repute, is as fine a specimen of athletic manhood as one could wish to see. He walked from Brighton to London recently to see his father in hospital and returning on foot, covered the distance, 105 miles, in twenty-three hours.

Of the other extra-muscular symptoms I shall say very little. Atrophy of the testicles, baldness, loss of body-weight, acrocyanosis and increased secretion of sweat, sputum or tears are most frequent.

Loss of the tendon reflexes is a very common sign. The knee-jerks for instance are often abolished even when the corresponding muscles show no evidence of disease. A ready explanation for this was found in the degeneration of the posterior columns described by Steinert in his case, which remained for many years the only one that had been examined after death, but this degeneration was absent in eight cases that have been examined recently, and I think that the reason for loss of the reflexes must be sought in changes in the muscles themselves.

Considerable attention has been paid to the psychological changes. These patients seem to succeed in arousing the ire of those who investigate them, and writers usually fill a couple of lines with unpleasant adjectives in attempting to describe the temperament and low mental capacity of their patients. Personally, except that they withhold the facts of the family history, that they discharge themselves from hospital before the examination is completed, that they promise to attend clinical meetings and fail to appear, and that they refuse quite politely but very firmly to render up a piece of muscle for microscopical examination, I have no fault to find with them.

We might have devoted the whole of our time to a consideration of these and other extra-muscular symptoms. I can only say now that no one of them is constant any more than myotonia or muscular atrophy is constant, but that in every case several of them will be found, and in no case will evidence be lacking to prove that dystrophia myotonica is something more than a purely muscular disease.

With these facts before us, what are we to say of the status of the disease? What is its relation in particular to Thomsen's disease and to the myopathies? I submit that it is entirely distinct from both. The separation from Thomsen's disease is easily made for there is nothing in common but myotonia, an occasional symptom in many unrelated diseases.

In the one myotonia is the symptom, in the other myotonia is but one of many symptoms, and it may be absent. In the one myotonia occurs in generation after generation (in five generations of Thomsen's own family); in the other it occurs in one generation alone, that is in the generation presenting the syndrome of dystrophia myotonica, but is absent in the preceding generations, although these show other signs of the hereditary disease, and in the generations that follow. The two diseases have never been found together in the same family, and muscular atrophy has never supervened in a patient with Thomsen's disease or in his offspring. Cataract and the other dystrophic symptoms are never found in the one; in the other they are always present. I need not labour this point. In 1911 Hirschfeld, Lewandowsky and Grund

stated in separate papers that dystrophia myotonica was completely independent of Thomsen's disease and I think we may follow them safely in company with everyone who has considered the matter since.

From the myopathies the separation is equally easy. In these, muscular atrophy is the symptom; it appears in many generations and cataract and dystrophic symptoms play no part. I leave for discussion, however, the many similarities between the two conditions, especially the nature of the muscular atrophy in both, and the possibility that the same kind of cause is active in both diseases.

If, then, dystrophia myotonica is a disease *sui generis*, what is its nature? Is it a disorder of ductless glands, a polyglandular syndrome? However much we may abhor the vague and baseless generalizations of some of those who write about the ductless glands, and however well we realize that the whole subject of the relation of these glands to the sympathetic and autonomic systems must soon be subjected to drastic revision, we are bound to admit that many of the facts support this hypothesis; many of the disturbances that I have grouped under the heading "dystrophic symptoms"—e.g., loss of sexual power, loss of hair, and so on—are known to occur in these disorders, and some of the other symptoms—e.g., Chvostek's sign, increased secretion of sputum, sweat and tears and the like, point to disturbances of sympathetic innervation. Cataract, too, perhaps must be included, for it is known to occur after removal of the thyroids and in other conditions such as tetany, where some defect of internal secretion seems to exist.

But willing as we may be, as we must be, I think, to agree that many of the symptoms are attributable to some defect of sympathetic and autonomic innervation, can we accept Naegeli's dictum that dystrophia myotonica is a pluriglandular syndrome, a disease of ductless glands pure and simple? I seek your advice, for myself the answer must be "no"! I base my decision on two points: First, the ductless glands were normal microscopically in two cases in which they were subjected to a special examination; and, secondly, there is no convincing evidence at present that myotonia and muscular atrophy can be caused by alterations in the internal secretions. This last statement I know leads us on to debatable ground, for the adherents of the pluriglandular hypothesis start out from the assumption that the functions of voluntary muscles *can* be influenced through the sympathetic and therefore by the ductless glands. Voluntary muscle, they say, has a double innervation, ordinary motor fibres supply the fibrils, the part of the muscle concerned in phasic contractions, whilst the sympathetic supplies the sarcoplasm, the part concerned with tonic contractions, and it is to disturbances in the sympathetic innervation of the sarcoplasm that myotonia and atrophy are said to be due.

This is not the time to enter upon a discussion of this alleged dual innervation of voluntary muscle, but as far-reaching conclusions are being based upon this notion, we should now remember that there is at present no real proof of it, and that the whole subject still remains within the realms of pure phantasy. I do not mean to deny that the myotonic disturbance *may* be due to changes in the sarcoplasm (myotonia is certainly myogenic in origin) nor to deny that the sarcoplasm may be innervated by the sympathetic, or that the sympathetic may have some trophic influence on striped muscle. I should indeed welcome any real proof of these, as they would supply a solution to many difficult problems in this and other diseases. All I mean is that the matter is still "not proven," and that in the present state of our knowledge we must look beyond the ductless glands for a complete and satisfying explanation of all the symptoms of our disease.

How are we to regard the muscular symptoms in relation to the general symptoms? That is the crux of the whole matter. The possibilities are three: (1) The muscular changes are primary and these changes give rise in some unknown way to the other symptoms; this has nothing to commend it. (2) The disturbances of sympathetic and autonomic innervation that cause the extra-muscular symptoms also cause myotonia and atrophy; we have considered this and rejected it. (3) The muscular and extra-muscular symptoms are to be regarded as co-ordinate, a common cause underlying them both; this, it seems to me, must be our standpoint at present.

But what is the common underlying factor? Is there a centre in the brain stem, in the hypothalamic region, in the tuber cinereum, as some have said, whence regulating influences pass to the autonomic and sympathetic systems and to the endocrine glands? Has this centre a trophic influence upon voluntary muscle? Is it here that we must look, as Curschmann has suggested, for some primary hereditary functional or morphological defect in this disease and in the myopathies perhaps as well?

A last word regarding cataract. I tread on unfamiliar ground but I understand that theories of the origin of senile cataract fall into two groups according to whether the changes in the lens are held to be primary and local, or secondary, the result of some general metabolic disorder. I gather, too, that whilst disorders of metabolism are accepted as obvious causal factors in the cataract of tetany, diabetes, nephritis, and thyroid disease, evidence is lacking at present to support the notion that general disorders play any part in the production of senile cataract.

But uncomplicated senile cataract in early generations of the families I have described was often the only evidence of a disease in which some abnormality of metabolism, perhaps some disorder in the control of internal secretion, seems to be the inherited factor. I must leave it for judges more competent than myself to decide whether the facts I have put before you have a bearing, not only on cataract in known disorders of internal secretion, but also on the larger and more difficult subject of senile cataract—in other words, on the whole problem of cataract in general.

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#### DISCUSSION.

Dr. WILFRED HARRIS (President) remarked that the anticipation of symptoms in hereditary disease was one of nature's methods of eliminating the disease. He would have liked to learn more of the condition of the children of patients suffering from dystrophia myotonica.

Dr. JAMES TAYLOR said that it was a remarkable fact that in many instances the families affected by hereditary disease were unusually large. He had known a family of ten children all of whom were myopathic, and had reported with Dr. Gordon Holmes similar large families with Leber's optic atrophy. Families who suffered from Friedreich's ataxia were also very often large. The relationship of dystrophia myotonica

to the myopathies was a question of great interest. He had once seen a case of the pseudo-hypertrophic type of myopathy in which there was cataract in one eye, but that was the only instance in which he had seen cataract associated with myopathy.

Mr. LESLIE PATON said that it was impossible to lay down any hard and fast rules as to the nature of the cataract associated with dystrophia myotonica. In the cases reported by Greenfield the type of cataract resembled that associated with retinitis pigmentosa, or with choroidal and ciliary degenerations and inflammations. It was a posterior cortical and not a posterior polar cataract. In a case which he had seen recently there was a ring of dotted granular opacities round the periphery of the lens associated with some peripheral striae like those seen in a senile cataract. It might be suggested that both the muscular changes and the cataract were associated with disturbance of the sympathetic system. In his experience, however, the form of cataract which accompanied dystrophia myotonica differed from that due to disease of the sympathetic system or superior cervical ganglion from the fact that he had never found in the former type any evidence of keratitis punctata, or of change of colour or loss of striation in the iris.

Sir JOHN HERBERT PARSONS, F.R.S., said that most cases which were called posterior polar cataract were in reality posterior cortical cataract, and these cases were often associated with general systemic disturbance. Too much attention had been paid in Germany to the very rare association of tetany with cataract.

Dr. F. M. R. WALSH said that he had recently had under his care a male patient, aged 30, who had been affected with dystrophia myotonica for five years, and had during that period developed minor epilepsy. He also had abductor paralysis of one vocal cord. He had complete foot-drop, with very little wasting of the anterior tibial group of muscles, and marked Rombergism.

Dr. GREENFIELD said he would like to give all the credit for the discovery of the association of dystrophia myotonica with hereditary cataract to the late Dr. F. E. Batten, who had discovered the presence of the disease in several members of the family which he recorded, and had helped greatly in the examination of the entire family. One member of this family showed a degeneration of the membrane of Bruch in one eye. Another had had some inflammation of both eyes which was cured in one eye by a proprietary ointment, whereas the other eye developed cataract. At the time of observation the patient (a female) had unilateral cataract.

Dr. ADIE (in reply) said that in many instances the families had gone down in the world, so that although the ancestors had been well to do, the family affected with dystrophia myotonica lived in poverty. In this and in other heredo-familial diseases the families were often large in the earlier generations and then dwindled in numbers. Many of those who suffered from dystrophia myotonica did not marry, nor had they any desire to marry, and some who married had no children. Some of the children were mentally defective, but so far as he knew none developed the muscular dystrophy. He asked whether cataract had been carefully looked for in other forms of myopathy. In its early stages a careful examination after dilatation of the pupil was necessary to reveal its presence. Epilepsy and various paralyses of the vocal cords had been described in this disease and Rombergism had been noted by Curschmann in one of his early cases which looked like tabes. Pains in the legs might be present in the early stages and add to the difficulty of diagnosis. Sudden giving way of the legs was common and complete foot-drop not uncommon, but he had not noticed that the weakness was out of proportion to the wasting. With regard to the morbid anatomy, although in Steinert's first case there was degeneration of the posterior columns, eight subsequent post-mortem examinations had revealed no such changes.

## Section of Neurology.<sup>1</sup>

President—Dr. WILFRED HARRIS.

### Case of Dystrophia Myotonica.

By W. J. ADIE, M.D.

W. A., MALE, aged 44. Married. Enjoyed good health until March, 1917, when, whilst undergoing military training, he passed through a severe feverish illness diagnosed as influenza; during convalescence he suffered from severe pains in the lower limbs; the pains subsided in a few months but left behind them a weakness of the lower limbs which steadily increased, and soon appeared in the arms as well. About a year after the onset of weakness in the legs, and some time after the arms became weak, he noticed that he had difficulty in relaxing his grasp; this trouble persists. His facial appearance has altered considerably; all his muscles have wasted; his speech is not so clear as it used to be; his hair is falling out rapidly; he has lost over 2 st. in weight; he has not had sexual connexion for some years; desire is absent. Apart from weakness in the limbs he feels well. He has never heard of a similar complaint or of cataract in any other member of his family. He has four healthy children.

Present condition: Facies myopathica; atrophy of sternomastoids, deep neck muscles, forearms and legs. Active myotonia in hand grasps. Reactive myotonia in tongue, small muscles of hands, deltoids and thighs. All tendon reflexes absent. Frontal baldness, acro-cyanosis, small soft testicles, narrow, high-arched palate.

### Case of Myotonia Congenita.

By LEWIS R. YEALLAND, M.D.

P. S., MALE, aged 18; one of twins. A paternal uncle suffers from cataract; nothing else from an hereditary point of view is discoverable. The patient was breast-fed and there was no difficulty in feeding him. He began to walk at the age of 2 years (a year later than his twin brother), was clumsy and made slow progress. At the age of 2½ his mother first noticed that when the patient's face was slapped for punishment, "his nose drew over to one side and remained there for a few seconds." At the age of 3, the patient was taken to India with his parents, and during his five years there nothing abnormal was observed; he walked normally and played games with other children. At the age of 8, he returned to England and began school. During his first week there (a) he saw double when looking at the blackboard from an angle; (b) his

<sup>1</sup> Cases shown at the Clinical Meeting held at the National Hospital for the Paralyzed and Epileptic, Queen Square, W.C., January 11, 1923.

## 46 Yealland: *Myotonia Congenita*; Collier: *Retraction of Eyelids*

hand became "stiff" when writing and he was unable to release his pencil from it; (c) at the first school drill he was compelled to fall out after twenty minutes participation, on account of a gradually increasing stiffness which began in the neck and spread rapidly to the arms, trunk and legs. The stiffness was superseded by limpness, and in less than thirty seconds from the commencement of the ictus he fell and was unable to move. He was taken home and remained in bed for three days limp and helpless but was able to swallow his food when fed. Similar attacks have occurred since, during exertion, and an attack will come on after thirty minutes to one hour's walk in the cold. In warm weather he is almost free from the attacks. After prolonged rest the patient is much better and he is in his best state the first thing in the morning after a prolonged rest. The condition is gradually becoming worse.

The patient is well developed. There are no deformities. Skin, mucous membrane, skull and spine, heart and lungs are all normal. The optic discs are clear and there are no evidences of organic changes in the central nervous system.

Repetition of any voluntary movement to order (e.g., eye movements, whistling, biting, yawning, flexion and extension of the elbow, wrist, knee or ankle), produces a gradually increasing tonic contraction of the part and will, if persevered in, result in complete cessation of movement. The same state of stiffness can be induced by the patient at will by throwing groups of muscles forcibly into contraction. Relaxation of muscles occurs gradually and is independent of the patient's will. Relaxation of the hand grasp is slow and awkward, the index finger extends first, afterwards the other fingers extend in succession and the movement seems to be dependent on the patient's effort. A slow tonic contraction of muscles follows a sharp tap of the percussion hammer. The myotonic electrical reactions are positive.

### DISCUSSION.

Dr. JAMES COLLIER said that there was no doubt about the diagnosis of myotonia congenita in this case. It showed in a typical manner the effect of cold in increasing the tendency to myotonia, and resembled most reported cases in the fact that the spasms were not decreased by exercise of the affected muscles.

Dr. WILFRED HARRIS (President) asked Dr. Yealland if he had seen attacks in which the patient was helpless. Was it possible that these attacks were in any way similar to attacks of family periodic paralysis?

Dr. YEALLAND (in reply) said that he had not himself observed such attacks, but the evidence he had been able to collect tended against this suggestion.

## Three Cases showing Retraction of the Eyelids.

By JAMES COLLIER, M.D.

(Shown by J. P. MARTIN, M.D.)

### (I) CASE OF TUMOUR AFFECTING THE MID-BRAIN.

THE patient presents the following signs: Pronounced psychic impairment. Papilloedema. The pupils react very poorly to light; often the right pupil does not react to light at all. Retraction of both upper eyelids. Defective upward movement of the eyes. Right facial weakness, especially on emotional movement. Absolute incontinence of urine. Slight weakness of the right side of the body, with extensor plantar reflex on that side.



## (II) CASE OF MID-BRAIN LESION (PROBABLY VASCULAR).

A. K. History: In October, 1921, the patient noticed shaking of the right hand; it passed off after about a fortnight. In February, 1922, the tremor returned; both the right arm and the right leg began to shake and she had double vision; her speech became slow and jerky.

Examination: Discs clear. Nystagmus: Vertical in right eye, oblique in left. Defective upward movement of both eyes; left eye turned slightly downwards and inwards. Ptosis of right upper lid; retraction of left upper lid with positive von Graefe sign. Speech very ataxic. During speech there is a rhythmical upward and downward movement of the eyebrows and tremor of the head. There is a coarse tremor of the right hand. No loss of power in the limbs. No changes in the reflexes, except that the plantar reflex on the left side is doubtful.

## (III) CASE OF DISSEMINATED SCLEROSIS WITH RETRACTION OF THE EYELIDS.

G. L., aged 41. Right leg began to drag seven years ago; since then symptoms steadily progressive—stiffness in both legs, spasms of lower limbs at night, precipitancy of micturition, incontinence.

Examination: Pupils equal: react normally. Nystagmus. Retracted lids, von Graefe's sign present. Slight intention tremor in hands: no ataxy or loss of power of upper limbs. Lower limbs very spastic, frequent flexion spasms.

Reflexes: Arm-jerks all present; knee- and ankle-jerks exaggerated. Abdominal reflexes absent; plantar reflexes both extensor. Cerebro-spinal fluid shows no abnormality.

## Case of "Spondylose Rhizo-mélique."

By JAMES COLLIER, M.D.

(Shown by J. P. MARTIN, M.D.)

PATIENT, a male. The vertebral column is rigidly ankylosed throughout its length; the hip-joints and knee-joints are also immobile; the right shoulder-joint shows great limitation of movement. The disease began ten years ago patient has been in a spinal carriage for five years. X-rays showed ossification of the short intervertebral ligaments.

## DISCUSSION.

Dr. J. G. GREENFIELD asked whether there was in this case any sign of gonorrhœa, or of any focus of chronic sepsis. Some cases of this affection had been attributed by French writers to gonorrhœa. He remembered a case under Dr. Batten's care in the National Hospital in 1911,<sup>1</sup> in which gonorrhœa was suspected but could not be proved. In the majority of cases the disease commenced about the age of 20, and this fact, as well as the nature of the pathological changes, and the distribution of the ankylosis (which always left the hands unaffected), distinguished spondylose rhizo-mélique from the chronic rheumatic diseases of the spine.

Dr. COLLIER (in reply) said that of six cases of this condition which he had observed none had suffered from gonorrhœa, nor was there any history or evidence of it

<sup>1</sup> Case shown before the Neurological Section on May 4, 1911. See *Proceedings*, 1911, iv (Sect. Neur.), p. 40.



## 48 Taylor: *Disseminated Sclerosis*; Walshe: *Case for Diagnosis*

in the present case. In one of his cases the disease came on after puerperal sepsis but in the others there was no focus of infection. In the majority of his cases the spine had been ventrally curved, but in one it was curved backwards as in the present case. In one case the affection started in the neck, and in six months spread down the spine to the sacrum, rendering the patient as helpless as if he had developed total paraplegia. He considered that the ankylosis of the spine was never bony as there was always pain on any attempts at passive movement.

### Case of Disseminated Sclerosis.

By JAMES TAYLOR, C.B.E., M.D.

H. C., AGED 47. Previous health good. Fracture of left leg below knee when aged 13. Present trouble started eight years ago by the left knee giving way and the left leg becoming weak. One year ago he discovered that on reading he "was using the left eye only." About the same date the left grasp became weak and a sensation of pins and needles was felt in the left forearm and hand. There was also dysuria.

On admission, November 6, 1922: Vision of right eye  $\frac{6}{18}$ , of left  $\frac{6}{60}$ . Left disc distinctly pale, chiefly in temporal half. Nystagmus on lateral deviation to both sides. Marked spasticity of left upper and lower extremities. Wasting of left trapezius and muscles of left shoulder girdle. Weakness of right rhomboids. The left arm and leg are less bulky and thinner than the right. Subjective sensation of pins and needles in left forearm and hand, and numb feeling in the feet. Slight diminution to cotton wool and pin-prick over left leg. Extensors wasted. Plantars: Right extensor, left flexor. Left ankle clonus. Knee- and ankle-jerks increased. Abdominals absent left, doubtful right. Wassermann reaction negative in blood and cerebro-spinal fluid. X-ray examination shows lipping and osteophytic outgrowths of cervical and lumbar vertebrae.

#### DISCUSSION.

Dr. JAMES TAYLOR said that the evidence of disseminated sclerosis in this case rested chiefly on the nystagmus, the difficulty in articulation and the signs of lateral sclerosis. The winging of the scapulae was probably due to some other cause, and it was possible that the osteophytic growths in the spine might account for this and some of the other symptoms.

Dr. COLLIER said that he would never admit wasting of muscles as a symptom of disseminated sclerosis.

Dr. WILFRED HARRIS (President) said that he had seen quite definite atrophy of the interosseous muscles of the hands in disseminated sclerosis.

### Case for Diagnosis: Spinal Compression or Disseminated Sclerosis.

By F. M. R. WALSH, M.D.

(Shown by W. G. WYLLIE, M.D.)

E. W., AGED 37. Past health good. Patient has six children, all in good health. Duration of symptoms eighteen months. Onset with pain in sole of left foot, followed by stiffness at back of left knee. In three months' time lost

the use of left leg. Sensation of pins and needles in left leg, when it was accidentally bumped. The right leg became weak and powerless two months later. Dysuria for the last six months.

On examination, December 27, 1922: No abnormality of the cranial nerves or upper extremities. Legs usually in rigid extension, feet plantar-flexed, halluces in extension. Marked adductor spasm. Flexor spasms of the legs have been becoming more frequent. No sensory changes. Knee- and ankle-jerks increased. Plantars give extensor responses. Abdominal reflexes: Upper feeble, lower absent. Reflex responses to cutaneous stimulation of both legs very active. Cerebro-spinal fluid clear, colourless, no cells; total protein 0.1 per cent. Wassermann reaction negative in blood and cerebro-spinal fluid.

Dr. C. P. SYMONDS said that he had never seen a case of disseminated sclerosis in which the protein in the cerebro-spinal fluid rose as high as 0.1 per cent.

### Case of Syringomyelia.

By S. A. KINNIER WILSON, M.D.

(Shown by Dr. W. G. WYLLIE, M.D.)

L. H., FEMALE, aged 16. Previous health: Diphtheria, "rheumatism" at the age of 4, and frequently since in legs, arms, and shoulders. Operation for adenoids when 10 years old. Twelve months' history of painless sores on the right hand and fingers.

November 12, 1922: Experienced a feeling of coldness in right half of body. At the same time she noticed that she could not appreciate the temperature of water with the right arm or hand. At the same period she noticed the left pupil was getting smaller and the left eyelid drooping.

On admission, December, 1922: Left pupil smaller than right, left eyelid drooping. Slight impairment of tactile sensibility, marked thermal and pain loss in left trigeminal area. Tongue on protrusion deviates slightly to the left. Very slight spasticity of right arm and leg. Right leg weaker than left. Sensory loss to pinprick and temperature over right half of body up to a transverse line in mid-cervical region. Faint diminution to tactile stimuli over the same area. Knee- and ankle-jerks greater on right than on left side. Plantars right (?), left flexor. Sphincters unaffected.

### Case for Diagnosis, possibly Amyotonia Congenita.

By S. A. KINNIER WILSON, M.D.

B. W., MALE, aged 11. Child born normally. Began to walk at normal age. He is the second youngest of eight children. At the age of 4 he was noticed by his mother to be double-jointed. He began at this time to "fall about." The weakness of the legs has been progressive in the last three years. At present he is unable to stand unsupported.

Mentally the child is bright and intelligent. When sitting up the back is unduly rounded. The arms are thin and their strength poor. The muscular power of the legs is very poor. Impaired muscle tone in all limbs, the deficiency being greater in the legs than in the arms, over-extension at the knees on passive movements being very prominent. No contractures present.

## 50 Wilson: *Congenital Neurosyphilis*; Howell: *Syringomyelia*

Arm-jerks very feeble. Abdominals active. Knee-jerks: Right greater than left, which is feeble. Ankle-jerks: Right feeble, left not obtained. Plantars: Right indefinite, left flexor. Sphincters unaffected.

### Congenital Neurosyphilis in Brother and Sister.

By S. A. KINNIER WILSON, M.D.

*Case I.*—D. J., MALE, aged 10. Epileptiform fits since age of 5. Automatism, spitefulness. Mentally clear, Hutchinson's teeth. Pupils unequal, poor light reaction. Wassermann reaction in blood, negative.

*Case II.*—G. J., FEMALE, aged 7. At the age of 5: paraplegia; unable to walk since arrest of mental development. Argyll Robertson pupils. Knee-jerks increased, plantars extensor, gait spastic, stamping. Ocular fundi, disseminated choroiditis. Wassermann reaction in blood positive.

#### DISCUSSION.

Dr. JAMES TAYLOR said that in his experience two members of a family of congenital syphilites usually suffered in different ways. One of his patients, a boy, afflicted with optic atrophy, was brought to hospital by a younger sister who had Hutchinsonian teeth, interstitial keratitis, and other signs of congenital syphilis, which were absent in the brother. The older patient eventually developed G.P.I. and died, whereas his sister remained free from symptoms of nervous disease.

Dr. WILFRED HARRIS (President) said that Dr. Taylor's cases argued strongly against the assumption that there was a neurotoxic strain of the *Spirocheta pallida*.

### Case of Syringomyelia, with much Sensory and Motor Impairment and little Wasting.

By C. M. HINDS HOWELL, M.D.

M. G., FEMALE, aged 23. Patient "fell down" in a seizure or faint while at work in February, 1919. Afterwards she had an illness thought to be influenza. After the illness she was troubled with "hot sweats" affecting the left shoulder and left side of neck and head. Later she noticed weakness of the left hand while at work. In February, 1922, the right leg began to feel stiff and she had a sensation of cold waves passing up and down this limb.

On examination it is found that the left pupil is greater and the left palpebral fissure smaller than on the right side. There is no nystagmus. There is partial loss of pain sense and of appreciation of cold over the left half of the forehead and over the lateral part of the left cheek. The left arm is weak, particularly in the hand grasp; the thenar and hypothenar eminences are wasted. The deep reflexes are absent in the left arm. The right arm shows no weakness or wasting. Both lower limbs are spastic, right more than the left. The knee- and ankle-jerks are exaggerated and ankle clonus is present. Both plantar reflexes are extensive. There is loss of tactile sensibility only over the outer side of the right leg. Pain sense is lost over both arms and both shoulders, and is impaired all down the right side of the trunk and over the whole of the right lower limb; on the left side of the body there is a similar impairment down to the lower border of the fourth dorsal segment both in

front and behind. Recognition of heat is completely lost over the whole of the right half of the body (except the face), and over the left arm and left side of the trunk down to the level of the tenth dorsal segment. Loss of recognition of cold corresponds almost exactly in its distribution to the impairment of sensibility to pain.

### Case for Diagnosis.

By C. M. HINDS HOWELL, M.D.

(Shown by J. P. MARTIN, M.D.)

G. S., MALE. History: In October, 1922, eight weeks before admission to hospital, patient's legs became weak while he was out walking; for about a week before that he had been losing the grip in the left hand and the left arm used to hang limply. Since the day when his legs gave way he has become steadily weaker and he has been aware of flickering in the muscles of his thighs. Since admission the right arm has become weak and he has become incontinent of urine and faeces. His neck has become stiff and it is painful when moved.

Examination: Left pupil slightly smaller than right. Fibrillation present in tongue. Complete flaccid palsy of left arm. Right arm becoming rapidly weaker. Abdominal and chest muscles weak; diaphragm does not seem to be acting. Left lower limb weaker than right. Fibrillation in muscles nearly all over the body, but especially in the thighs; wasting most pronounced in the muscles of the back and in the left lower limb. Sensory loss variable; fairly complete in both arms, less complete on chest with a level at the angle of the sternum. Sensory loss on the lower limbs much less than on the upper; right lower limb more affected than left. Reflexes: Knee-jerks brisk; plantars extensor.

### DISCUSSION.

Dr. MARTIN added that most of the signs presented by this case suggested the diagnosis of compression of the cervical region of the cord by a tumour. But this would not explain the fibrillation which was present. X-rays showed erosion of the second cervical vertebra and some shadows suggesting an intrathoracic tumour. As the condition was rapidly progressive he considered that it was probably due to a diffuse sarcomatous or gliomatous invasion of the spinal cord. The cerebro-spinal fluid contained 0.1 per cent. protein.

Dr. WILFRED HARRIS (President) described a somewhat similar case, with total paralysis and anesthesia of the right arm and partial paraplegia, in which he had advised operation. The surgeon found no tumour but noted no pulsations of the cord or dura mater. He therefore explored upwards under the lamina arches and came against a resistant mass. When he tried to break through this there was a gush of blood which proved, post mortem, to have come from an aneurysm of the posterior inferior cerebellar artery.

Dr. JAMES TAYLOR said that he had seen a similar case, which also presented the symptom of fibrillation of muscles, and which proved to be due to gliomatosis of the cord.

Dr. G. RIDDOCH described a case of quadriplegia, with signs of spinal compression and multiple root lesions but with few sensory alterations, due to sarcomatosis of the meninges. The cerebro-spinal fluid in this case was yellow and very highly albuminous and contained many tumour cells some of which showed mitotic figures.

**Case of Disseminated Sclerosis.**

By W. ALDREN TURNER, C.B., M.D.

G. W., MALE, aged 35. About one and a half years ago patient began to be troubled with a trembling of the right arm, becoming more marked when he used the arm; this tremor has gradually become coarser. About the time the tremor commenced patient's gait became somewhat unsteady. Fifteen months ago his speech became jerky and has got steadily worse up to the present. Four years ago patient fell off a bicycle, striking his head and bruising his right elbow.

Examination: Discs normal. Pupils unequal, left greater than right, regular, slightly eccentric, with some retraction of eyelids; they react normally. External ocular movements good, no nystagmus, squint or diplopia. Movements of face, palate and tongue normal. Numbness in both hands. No demonstrable loss to pinprick or cotton wool. Sense of position in space and localization of touch normal. No astereognosis. Enormous intention tremor of the right arm, so great that it shakes the whole body. The tremor is not purely intentional, for it is sometimes present mildly in the shoulder during rest. The left arm shows a slight tremor of the intention type. There is no loss of power or limitation of movement. Arm-jerks: Equal on the two sides; biceps-jerk not obtained. Knee- and ankle-jerks: Right greater than left. No ankle clonus. Abdominals: Present and equal on the two sides. Plantars: Both flexor. Gross ataxia of gait: The arms, especially the right, make large movements which react on the whole body. No Rombergism. Wassermann reaction negative. The cerebro-spinal fluid is normal except that it gives a "paretic" curve with the Lange test.

Progress: Little change noticed in arms, though patient feels that the tremor of the right arm now involves the shoulder in greater degree. Both legs are affected by tremor and the left is now rather more affected than the right.

**DISCUSSION.**

Dr. C. P. SYMONDS said that he had noticed in the history of this case that the patient had had an illness called "influenza" six months before the onset of the tremor. He considered it possible that this was really a mild attack of encephalitis lethargica. He had seen involuntary movements follow an attack of encephalitis lethargica so mild as not to keep the patient in bed. He (Dr. Symonds) had observed Lange reactions of the "paretic" type with the cerebro-spinal fluid of two cases of encephalitis lethargica.

Dr. G. RIDDOCH said he was inclined to agree with Dr. Symonds in considering that this case was due to encephalitis lethargica. The steady onset of symptoms without any remission was unlike disseminated sclerosis. He had often noticed similar gross involuntary movements coming on gradually after encephalitis lethargica.

**Unilateral Affection of Cranial Nerves, 7, 9, 10, 11 and 12.**

By C. P. SYMONDS, M.D.

MISS T., aged 30. In July, 1919, she had some teeth extracted. Shortly afterwards she noticed a discharge from her left ear, for which she did not consult a doctor. This was followed by gradually increasing deafness in the

left ear. In March, 1920, she first noticed hoarseness, which prevented her singing. About the same time she began to experience pain in the left shoulder and found that this occurred especially when she attempted to carry heavy objects in the left hand. In the summer of 1921 her voice improved, so that she was again able to recite, though singing was still impossible. The pain in the left shoulder was also less noticeable. During the last year the voice has again become hoarser, and the trouble in carrying things with the left arm has increased. She has also occasionally noticed some difficulty in swallowing. In October, 1922, for the first time she had a brief attack of left facial spasm. During this the eye closed and the whole side of the face was screwed up. Since then she has had several such attacks.

On examination the positive findings are: Left-sided conduction deafness together with a fleshy mass obscuring the membrana tympani, ? a polyp. A fragment has been removed for microscopical examination; this shows it to be a capillary angioma. Left facial palsy of peripheral type. Paresis of the left vocal cord and left side of the palate. Weakness and wasting of the left sterno-mastoid and trapezius. Atrophic palsy of the left side of the tongue. Blood: Wassermann reaction negative. X-ray appearances of head and upper cervical spine negative.

*Postscript.*—The left mastoid antrum has been explored by Mr. Mollison and was found healthy.

### Case of Unilateral Affection of Cranial Nerves 9—12 (Tapia's Syndrome) associated with Chronic Otitis Media.

By C. P. SYMONDS, M.D.

MISS C., aged 35. She has suffered from bilateral otorrhœa from time to time since childhood. She had tonsillitis at the age of 28, is subject to headaches, and has attended Guy's Hospital for constipation. In February, 1921, she first noticed hoarseness, and was referred from the medical out-patients' department, where she was attending for her constipation, to the Throat Department. It was there observed that the left vocal cord was paralysed. When seen a few days later the positive findings were: Paralysis of left vocal cord and weakness of left side of soft palate. Weakness and wasting of left sterno-mastoid and trapezius. Atrophic palsy of the left half of the tongue. Evidence of old standing otitis media on both sides with a moderate degree of conduction deafness. The blood: Wassermann reaction negative. X-ray plates of the head and of the upper cervical spine showed no abnormality.

Under observation this patient has shown little change during the past two years except for the tendency of the hoarseness to diminish.

#### DISCUSSION.

Dr. WILFRED HARRIS (President) considered Dr. Symonds' cases to be similar to those described in text-books as "polyneuritis cranialis," the causation of which had been traced in some cases to otitis media.

Dr. J. COLLIER said that he had followed seven similar cases to autopsy and in every case osteo-myelitis of the base of the skull had been found. In one the disease had followed a bullet wound of the cranium, in another a tubercular osteomyelitis of the

basisphenoid, and in the remainder ear disease was the cause. The appearance seen in the posterior fossa at autopsy was a plum-coloured cushion of raised and thickened dura mater with disintegration of the underlying bone. In every case the causative organism was a streptococcus. The long duration and the absence of pyrexia were remarkable. In one case there was no clinical evidence of otitis media, although the autopsy proved that this had existed. He placed no reliance on X-ray evidence in this condition as it was very difficult to get satisfactory radiograms of the posterior cranial fossa. The cause of death had been lepto-meningitis in each of his cases excepting the tubercular case, in which the carotid artery was eroded and had burst into the pharynx. In one case a hemiplegia had developed and was found to be due to thrombosis of the superficial veins of the brain. Replying to a question by Dr. Symonds as to whether the operative findings in the first of his two cases were not against his conception of the disease, Dr. Collier replied that in two cases following ear disease, autopsy showed that the mastoid region was healthy but that the infection had spread through the apex of the petrous bone.



## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### Disorders of Function in the Neurone.

By E. D. ADRIAN, M.D.

THIS paper attempts to show how a knowledge of the physiology of nervous conduction may help to explain what is happening when the activity of the neurone is impaired by disease. It is true that in many nervous diseases the familiar symptoms are due not to the defective activity but to the complete failure or death of a group of neurones in some part of the central nervous system, and our treatment must depend on a knowledge of bacteriology, of the paths of infection, the pathology of blood-vessels, &c.

There are, however, some diseases in which the symptoms appear to be due to a true disorder of function such as a diminished power of conduction, or an excessive response to a stimulus. Such disorders as toxic polyneuritis or trigeminal neuralgia are perhaps the best examples, and here an exact knowledge of what is wrong with the neurone would become a matter of practical value.

It cannot be claimed that our present knowledge of the physiology of the nervous impulse does give very much help in understanding these disorders, but at least it gives an indication of what is possible and what is not.

Our knowledge of nervous conduction is based mainly on a study of the effects of electrical stimuli on isolated nerves. The disturbance which travels down the nerve after an adequate stimulus reveals itself by three changes: (a) There is a momentary change of electric potential which passes down the nerve; (b) there are changes of excitability which follow in the wake of this; and (c) there is a reaction in the organ to which the nerve is connected.

By combining these different indicators it can be shown that the electric response is an invariable accompaniment of the impulse, and that it forms the only measurable expenditure of energy occurring in its transmission. The electric response must be caused by a movement of ions in or out of the fibre in the active region, and this movement of ions seems to be all or almost all that happens.

The exact mechanism of the change is naturally a more debateable matter, but there is a great deal of evidence to support what is generally known as the "membrane theory," proposed originally by Bernstein and advocated very

strongly in recent years by Lillie [1]. There is no need to go into this at any length. It supposes that the electric response is caused by a leakage of ions stored up under pressure inside the fibre; that these ions can leak out because in the active region there is a sudden increase in the permeability of the surface layers of the fibre; and that the movement of ions in one section causes a similar change in the next section, so that the effect is propagated rapidly down the fibre. An important point about this theory is that the energy needed for the transmission of the impulse is derived, not from the stimulus, but from the fibre itself, and that it is extremely small. Actual measurements of the electrical effect and the absence of any temperature change have shown that the expenditure of energy for each impulse is less than one hundred thousandth of that expended in the twitch of a muscle fibre [2]. Evidently, therefore, the transmission of nervous impulses does not make any great demands on the metabolism of the cell as far as mere calories are concerned. The other important point is that the fibre, or some part of it, is supposed to be surrounded by a membrane or surface layer which is in a state of very delicate equilibrium. Both the appearance and disappearance of the active state (or the "impulse") are supposed to depend on a change in the permeability of the surface layer, and it is absolutely essential that this layer should react rapidly and completely to a small change in its surroundings [3]. For what follows it is not necessary to accept the membrane theory; it is mentioned because as far as its general outlines are concerned it rests on a fairly sure foundation and it gives a definite picture of the nervous impulse as a little patch of surface leakage spreading along the fibre and being sealed up again almost as soon as it has formed.

Whatever its precise nature may be, the nervous impulse is a disturbance which never lasts for more than a few thousandths of a second at any point. The period of activity dies away, and is succeeded by a refractory period during which the fibre cannot be made to respond at all. The refractory period is important, for it shows that the nerve fibre cannot remain continuously active; it can only respond intermittently—like a beating heart—and in a mammalian nerve trunk the impulses can succeed one another at a rate of 400 a second, but they can never become fused. It is, however, quite likely that their effects can become piled up at synapses or nerve-endings. The existence of the refractory state has often been taken to show that the nerve fibre is fatigued after the passage of an impulse, and needs time to recuperate. This suggests that we might find considerable changes in the duration of the refractory state when the neurone is diseased, that it might take a longer time to recover after each impulse. But it is an interesting fact that under experimental conditions the only circumstance which seems to affect the refractory period at all is a change of temperature. Lucas [4] showed that the conductivity of the nerve could be completely abolished by alcohol without any alteration in the refractory period, and there is no evidence that other narcotics or other conditions which impair the activity of the fibre have any appreciable effect on the rate of recovery from an impulse. This generalization is probably too sweeping, but it shows that we need not expect much alteration in the refractory period of a diseased neurone.

Now impulses of the kind we have described, electric disturbances followed by the refractory state, are the only kind of change we can detect in an isolated motor or sensory nerve trunk, but it does not follow that some other kind of change may not occur in the nerves when we are dealing with the intact central nervous system. We have no direct evidence about the kind of

disturbance which travels up a sensory nerve when the end organs are stimulated, though the fact that sensations and reflexes are given by the electrical stimulation of a sensory nerve trunk shows that the impulses set up by electrical stimulation of a nerve fibre cannot differ very much from those occurring naturally. But there is fairly good evidence that the skeletal muscles are normally brought into play by impulses coming out of the central nervous system which do not differ in any way from those studied in isolated nerve. The most direct evidence is that supplied by Gasser and Newcomer [5]. Using a valve amplifying device with the string galvanometer, they have been able to record the electric responses in the phrenic nerve during respiration, and they find that each contraction of the diaphragm is produced by a series of about 80 to 180 impulses of the usual type occurring at the rate of 70 to 100 a second. A good deal of less direct evidence comes out of work on the electromyogram and Hoffmann's recent studies on the tendon reflexes [6]. The dispute about tonic contraction, sympathetic innervation, and so on, is still with us, so that we cannot exclude the possibility that there may be other kinds of nervous activity, but at any rate a good deal of the work of the nervous system is done by impulses of the ordinary type.<sup>1</sup> It is possible, therefore, to analyse a little more closely the nature of some of the disturbances of function which may be produced experimentally or by disease.

The simplest disturbance, and one which accounts for a great deal of the symptom production of nervous disease, is a partial or a complete failure to transmit impulses. Now in an isolated nerve trunk we can bring about a partial or complete failure of conduction in many ways—by narcotics, poisons, mechanical injury, &c.—but however we do it, the process of failure always seems to be the same. When it reaches the affected area the impulse becomes smaller and smaller as it travels along until eventually it dies out altogether. The greater the degree of narcosis, the more severe the injury, the more rapidly will the impulse die away and the shorter the distance will it be able to travel before it is extinguished.

This disordered form of conduction, "conduction with a decrement" as it is called, is always the first step towards complete failure however this may be brought about. In one sense we may regard it as merely a reversion to a more primitive type of conduction. Verworn [7] pointed out ten years ago that the transmission of an impulse down a highly specialized structure like the medullated nerve fibre is a much more efficient process than that taking place in less specialized tissues. In the long cell processes of some of the protozoa, and in the nerve network of the sea anemone, conduction with a decrement seems to be the normal event: the disturbance set up by a stimulus becomes less and less intense as it travels away from its point of origin and the initial intensity of the disturbance varies with the strength of the stimulus, so that the disturbance set up by a strong stimulus will travel further and cause a more widespread response. This kind of conduction is all very well in an animal with a diffuse nervous system, but it would be very awkward in animals in which there is a central nervous system joined to the end organs by nerve trunks of different length. At any rate in all the more highly developed nervous systems we find that in the specialized conducting tracts (the peripheral nerves and white matter) the disturbance is normally conducted over long distances without any reduction in its intensity, so that it does not matter how far the end organ may be from the central nervous system. This

<sup>1</sup> Recently Brücke has given a definite proof that reflex inhibition is also produced by impulses of this kind, *Zeitschr. f. Biol.*, 1922, lxxvii, p. 29.

more perfect form of conduction carries with it one possible disadvantage, namely, that the intensity of the disturbance (or impulse) must always be the same ; it can no longer vary with the strength of the stimulus which sets it up. The all-or-nothing reaction of the nerve fibre excludes one possible method of varying the intensity of response in the end organ, but it seems to be a necessary consequence if the impulse is to be conducted without loss of intensity, and there are other ways in which a graded response may be obtained. But this highly developed form of conduction with impulses of maximum and unchanging intensity can only go on as long as the fibre is in a healthy state and as soon as this is interfered with the fibre reverts to the less perfect form of conduction and the impulse begins to dwindle in intensity as it passes through the unhealthy area. If the length of this is considerable or the disorder severe, the impulse will be extinguished altogether and there will be a complete failure of conduction. The immediate cause of the change from the normal to the decremental type of conduction seems to be an interference with the surface membrane ; narcotics make the surface less permeable [8], apparently by dissolving in it and causing a mechanical obstruction ; injury may destroy it altogether, but anything which will interfere with its sensitiveness to changes in ionic concentration will also prevent the proper development of the electric response and its transmission from one section to the next. Incidentally the impulse is usually conducted at a slower rate in the affected region.

The practical bearing of all this is that any condition which interferes with the conductivity of a nerve fibre will have a greater effect in proportion to the length of nerve over which it acts, for the decrease in the size of the impulse is proportional to the distance it has to travel in the affected fibre.

There is not very much to be said about mechanical injury or pressure as a cause of imperfect conduction. A good deal of research has been done on the effects of gradual compression, which can abolish conduction temporarily without leading to any permanent impairment, and it has been shown that moderate pressure over a considerable length of nerve has the same effect as greater pressure over a shorter length, which is what we should expect if the compression produced decremental conduction.

Much more interesting is the failure or partial failure of conduction which occurs in the various forms of toxic neuritis or polyneuritis, due to arsenic, alcohol, diabetes, diphtheria, and so on. Here the whole length of the nerve trunks must be exposed to the toxin and from what we know of nervous conduction it seems almost certain that the first sign of failure will be the appearance of conduction with a decrement, the gradual dwindling in the intensity of the impulse as it passes along. Now if all the nerves were equally affected and uniformly affected throughout their length, we should find that the first impulses which would fail to reach their destination would be those which had the longest distance to travel. In other words we should find that the paralysis and the loss of sensation would appear first in the hands and feet and would rarely affect the trunk. No doubt there are other factors as well to account for the distribution of the symptoms : for instance the end of a very long axon may be less resistant because it is a long way from the cell nucleus, and the fact that the histological changes are often confined to the extremities of the nerve trunks makes it clear that the long fibres are more seriously affected than the short. At the same time the great probability of decremental conduction is a factor which must certainly be taken into account : it would lead inevitably to an earlier failure in the longer conducting paths and this must play some part in the distribution of the symptoms.

In the polyneuritis produced in birds by feeding them on polished rice there is definite evidence of decremental conduction in the nerves. Kato, Shizume and Maki [9] have made some very interesting observations on muscle nerve preparations from chickens fed on polished rice and they have shown that the affected nerves can conduct an impulse for a short distance but not for a long, and that the velocity of conduction is slower than normal. The disordered conduction clears up in a few hours after the injection of rice-bran extract, but there is no general agreement as to how the extract works.

Unfortunately, decremental conduction is such a universal reaction to any harmful agent that its existence does not help us to decide the mode of action of any particular toxin. To go any further would need an investigation of many other properties of the nerve fibre and would involve the removal of the nerve from the body. It is true that there is one property, that of excitability to electrical stimuli, which can be determined in an intact subject, and this function can now be investigated more thoroughly by determining a factor which relates the strength and the duration of the current needed to stimulate. Lapicque, who has inspired most of this work, has called this factor the "chronaxie;" roughly speaking, it gives an idea of the rate at which the different reactions take place when the nerve is stimulated. Laugier and Bourguignon have measured the chronaxie in different nervous diseases and found distinct though not very large changes in it, but, again, there is not much to separate the effects of different toxins.

So far we have confined ourselves to a disorder of function in which the activity of the neurone is diminished. It would be of greater value to know something of the disorders in which there is not a failure but an excess of activity, disorders like trigeminal neuralgia for instance, or epilepsy. Unfortunately information derived from a study of medullated nerve fibres cannot be applied without qualification to cover the fibres which are concerned with pain sensations and still less can we apply it to the cells in the cortex. At the same time there can be very little doubt that in disorders of this sort the neurone may suddenly start discharging a series of impulses in response to a stimulus which would normally have little effect on it. Here we can at least get a clearer idea of what may be wrong by considering the normal process of excitation.

When an electric or mechanical stimulus is applied to a nerve fibre it will not be successful in setting up an impulse unless it satisfies two conditions: (a) It must be able to produce a disturbance (a change in polarization) of a certain minimum intensity in the fibre; and (b) it must do this in a certain definite time. The nerve appears to adapt itself rapidly to the new conditions, so that if the stimulating current is increased gradually from zero, instead of being turned on suddenly, it may never stimulate at all, although its final value is very large. This power of adaptation plays a very important part in the activity of the nerve fibre, for as long as it is effective an impulse will only be set up when the external conditions are actually changing. As soon as a steady state is reached, e.g., during the passage of a constant current, or after an injury has been established, the fibre should come into equilibrium with its surroundings again and no more impulses should be set up. The power of adaptation is limited—a very strong constant current may set up a series of impulses—and it varies to some extent with the state of the fibre. We know at least that it is altered by a change in the relative proportions of the different ions in the perfusing fluid, but physiologists have not studied it at all

exhaustively. It is pure speculation to suggest that the excessive response of a neurone may be due to a failure of this adaptive mechanism, but if it did fail the neurone would certainly respond with a long series of impulses to stimuli, such as a steady mechanical pressure, which would normally be quite inadequate to produce any response. On the other hand the adaptive mechanism may be normal and the trouble might lie in an abnormal instability of the surface membrane, which would become "active" in response to an unusually small change of conditions. To decide between these alternatives, if they are alternatives, is not beyond the range of experiment, but this would only be a beginning, and before we could get very far we should have to learn a great deal more about the mechanism of pain in terms of the nervous impulse. Here we are still very much in the dark, in spite of the great insight into the central mechanisms of pain which Dr. Head's work has given us.

To go any further with these speculations would lead us into fields where our present knowledge of the nervous impulse would be of very little use. Knowledge as to what happens in the synaptic areas of the central nervous system is still too uncertain to supply us with any basis for a study of disordered conduction there. At the same time we have recently seen what great advances may be made in the understanding of cardiac disorders by means of detailed investigation of the development and conduction of the impulse in the heart muscle. So far as we can tell the change which is conducted in the heart differs only in degree from the impulse in a nerve. The conducting paths in the nervous system are much more complicated than in the heart, but that is perhaps all the more reason for considering the results which may follow when the excitation or conduction of the nervous impulse is modified by disease.

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## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### DISCUSSION ON THE TREATMENT OF NEURO-SYPHILIS.

Dr. E. FARQUHAR BUZZARD.

IT may have been an accident, but it is certainly not inappropriate that this week should have been chosen for a discussion on the treatment of neuro-syphilis. Four hundred and thirty years ago last Sunday Columbus and his companions landed in Europe on their return from their first voyage to America, bringing with them, as an undiscovered stowaway, the spirochæte which was destined to play such havoc with the European nervous system. The choice of date for this discussion must have been accidental, as our honorary secretaries would not have risked attention being drawn to the fact that the responsibility for the appearance of syphilis in our isles lies in the first instance on Aberdeen and in the second on Edinburgh, whence the disease spread rapidly to other parts of the United Kingdom. It is only fair to note, however, that these two cities have never shirked their responsibility in the matter, supplying, as they have done, innumerable and valiant recruits to the ranks of the medical profession in its crusade against the scourge, recruits who have followed the enemy into every corner of the country.

The privilege of opening a discussion of this kind is more or less bound up with the duty of provoking controversy, and I hope to fulfil the latter by making a general attack on the profession, to which I have the honour to belong, on its conduct of the crusade against syphilis from the time when ignorance afforded a reasonable excuse for inefficiency, until the present day when it ought to know better.

In order to simplify the issues, I propose to lay down dogmatically certain principles to which I have no doubt some of you will take exception.

In the first place, no distinction can be drawn between the interstitial and parenchymatous varieties of neuro-syphilis in so far as the principle governing their preventive and curative treatment is concerned; to my mind they both afford evidence of an active process in the nervous system provoked by the presence of the *Spirochæta pallida*.

In the second place, the prevention of neuro-syphilis must depend on the complete eradication of the spirochæte from the bodies of infected persons or, alternatively, the continued abeyance of its activities. Although the first desired result has no doubt often been attained by treatment, we have never had, and we are still without, reliable data on which to base the opinion that any particular individual has been cured of syphilis.



Thirdly, if these two statements are accepted, we are never justified in assuring a patient that he is cured of syphilis even if ten, twenty or thirty years without symptoms or signs of the disease being manifest have elapsed since the date of infection. On the contrary, it is our duty to advise all patients who have been infected to continue with intermittent treatment for the rest of their lives.

These propositions deserve more critical consideration in their proper order.

Many of us remember the time when it was generally taught that while the interstitial form of cerebro-spinal syphilis could be regarded as the product of the active virus, the parenchymatous or so-called para-syphilitic forms were manifestations of a mysterious after-effect ofluetie infection. Much controversy raged around this hypothesis, and it was even maintained by some that tabes and general paralysis could be more fairly attributed to the prevailing methods of treating syphilis than to the disease itself. The discovery of the spirochæte in the central nervous system brought about a sudden and complete change of opinion, which now favours the view that both tabes and general paralysis are produced by active syphilization of the nervous tissues. If this view is correct we must assume that the principles governing their prevention and curative treatment are essentially the same as those we recognize in relation to cerebro-spinal syphilis and indeed to all late manifestations of syphilis in other organs. In other words, the appearance of neuro-syphilis in any form must be admitted as evidence of failure in our attempts to cure primary syphilis.

What has the profession done about it? Admittedly, its armament against syphilis has been added to. Various metals besides mercury have been brought into the fray, but the same blindness to the teachings of clinical experience is still displayed.

The cure of syphilis by two years of mercury was replaced first of all by one dose of salvarsan, and later by almost daily increments in the number of doses of that and of other drugs. But late manifestations of syphilis are still frequent. I submit that we shall never obtain better results until we frankly admit that we cannot justifiably use the word "cure" in relation to syphilis, and unreservedly support the principle of life-long treatment. The following quotation from an article on syphilis in one of our most recent text-books illustrates the fallacy underlying present-day teaching:—

"The main principles to be observed in the treatment of syphilis are: (1) To begin as early as possible, before the parasite has become buried in the sclerosed primary sore or entrenched in comparatively inaccessible regions, such as the central nervous system; (2) to continue as long as experience shows that there is a possibility of the patient relapsing if treatment ceases."

I do not quarrel with these exhortations, but the second loses its force when followed a little later by this sentence:—

"There is evidence to show that, judging by the persistence of a negative Wassermann reaction for some years afterwards, and the propagation of healthy children, many cases are cured by a comparatively short course of arsenobenzol and mercurial injections, whilst others, apparently the same in character, have relapsed again and again after the same course."

Is it fair to ourselves or to our patients to judge by the persistence of a negative Wassermann reaction and the propagation of healthy children? Clinical experience is full of evidence to the contrary. Probably the majority of our

tabetic and general paralytic patients have propagated healthy children, and certainly large numbers have negative Wassermann reactions while displaying definite clinical evidence of these diseases. I submit that despite its undoubted scientific interest the Wassermann reaction is an obstacle rather than a help in the practice of medicine. A positive reaction is sometimes a valuable piece of confirmatory evidence. A negative reaction is almost without value as evidence, but unfortunately has become so highly credited that it is a direct menace to proper treatment. It gives rise to delusions of security in the minds of doctor and patient, satisfying the desire unconsciously cherished by most of us to shift our responsibility on to some test.

You will have gathered that I regard continuity of antisyphilitic therapeutics as the foremost principle to be observed in regard to both the prevention and the treatment of neuro-syphilis. That periodic courses of treatment year after year will prevent the development of neuro-syphilis has yet to be proved, but it is significant that patients who neglect the treatment of the primary infection often develop nervous manifestations at an early date, and that in those patients who are subjected to thorough treatment for several years to begin with, the cerebral and spinal complications are often long delayed.

Many years ago Fournier tabulated 321 cases of tabes with previous syphilis in the following way: There were 24 in which there was no treatment at all, 70 in which there had been absolutely insufficient treatment, 108 in which three to six months of mercury had been given, 74 in which treatment had lasted six to twelve months, 32 in which it had lasted one to two years, and only 13 in which it lasted two to four years. These figures are significant in themselves, but it would be far more interesting and instructive to know in what percentage of cases of syphilis treated intermittently year after year tabes, general paralysis, or other forms of neuro-syphilis would develop.

For many years it has been recognized that meningeal and vascular lesions respond to antisyphilitic measures, but it is not long since neurological authorities denied that any benefit could be derived from treating tabetic or general paralytic patients on similar lines. Personally I am convinced that this view was mistaken, and I have brought evidence before this Section showing that in regard to tabes, at any rate, the combination of early diagnosis and continued treatment is attended by most satisfactory results. The question of general paralysis is somewhat different, although I am not altogether without hope that with sufficiently early and sufficiently prolonged treatment better results may be obtained than those attending other measures.

It need hardly be said that, in speaking of the beneficial results of treating neuro-syphilis by any particular method, it is not suggested that it is possible to do away with such physical signs as Argyll-Robertson pupils, areas of analgesia and absent tendon-jerks. If the progress of the disease can be arrested and disorders of function can be improved or cured by re-educational measures, it is all that one can expect.

We shall probably find, in the course of our discussion, that there is much difference of opinion in regard to the efficacy of various methods of treatment. There seems to be a general feeling that, in spite of the good results often obtained by the administration of arsenical compounds, mercury ought not to be discarded, and personally I am in the habit of using both metals in the initial treatment of neuro-syphilis. The ideal route for the administration of arsenic is still undecided, but I hope to learn much in regard

to this important subject to-night. I have never been enamoured of intrathecal medication, partly because I have seen no results following this method which are unobtainable by intravenous or intramuscular injections, and partly because repeated lumbar punctures are a source not only of much distress but also of some danger to the patient.

We may expect to hear that antisyphilitic treatment should be guided by repeated examinations of the blood and cerebro-spinal fluid. For my part, I prefer to continue with periodic treatment without paying any attention to the blood Wassermann reaction in patients suffering from tabes or who have suffered from interstitial lesions in the brain or spinal cord. On the other hand, occasional examination of the cytology of the cerebro-spinal fluid may be helpful in regulating the amount of treatment desirable in the early stages of acute conditions.

Another question of interest concerns the contra-indications to antisyphilitic treatment. We shall probably all agree that great caution is required with patients suffering from advanced cardiovascular changes, especially in relation to intravenous injections. Albuminuria is not in itself a contra-indication to mercury or arsenic, and it will often clear up during the administration of these drugs. In other words, renal inefficiency, rather than albuminuria, must be regarded as the contra-indication.

It used to be stated that mercury and arsenic should not be given to patients suffering from optic atrophy, and at one time I shared the gloomy view generally taken in relation to the prognosis of this condition. Latterly, however, I have had reason to modify this opinion, having seen a few cases of early optic atrophy treated energetically with mercury and arsenic, and apparently deriving great benefit. In this connexion I was much interested in an article written by Leslie Paton on tabes and optic atrophy.<sup>1</sup> He supplies information suggesting why it is that some forms of atrophy of the optic nerve are more amenable to treatment than others.

My remarks have been confined to the treatment of neuro-syphilis by antisyphilitic therapy, but in the course of the evening I hope we may hear something of other methods. There is, for instance, the treatment of general paralysis by inoculations of malaria or other infective agents, of which I have read, but of which I have had no personal experience. Whether this hope is realized or not, I am satisfied that there is ample matter for profitable discussion in the principles governing antisyphilitic therapeutics and in the technique which should be employed to ensure its success.

#### Dr. H. MACCORMAC, C.B.E.

Although little qualified to discuss a subject demanding considerable technical knowledge of neurology, as a syphilologist, however, I have the opportunity of seeing syphilitic disease in its various forms and in this way may have become familiar with certain general principles upon which to base methods of treatment.

The general study of syphilis may be undertaken in two different ways: Either attention may be paid to the signs and symptoms, their nature, and the effect of treatment on them; or observation may be confined to the biological reactions, such as the Wassermann reaction. Each of these methods has its special purpose and they are to some extent complementary, but there is

<sup>1</sup> *Brit. Journ. Ophth.*, 1922, pp. 289-316.

a real distinction between them, since the former is concerned with the anatomical structures involved, while the latter is an indication of infection or freedom from infection by the *Spirochæta pallida*. This distinction should be noted because although many forms of treatment may remove the lesions, unless we can permanently cause the biological reactions to become "negative" we are not entitled to assume that a cure has been effected.

When we investigate syphilis in the latter direction, that is, in respect of the biological reactions, we find a considerable difference in the effect of treatment in the early and late periods. In the early periods the Wassermann reaction may be profoundly and rapidly influenced by intensive treatment. This is well shown when systematic observations are made in the different stages of the disease. The following are examples taken from early stages to illustrate this point:—

EARLY SYPHILIS: WEEKLY OBSERVATIONS OF THE WASSERMANN REACTION IN CASES UNDER INTENSIVE TREATMENT.

(a) Primary Syphilis.									
1	2	3	4	5	6	7	8	9	10 weeks
++	++	+	+	—	—	—	—	...	...
—	...	++	+	—	—	—	—	—	—
++	++	++	+	—	—	—	—	—	...
(b) Secondary Syphilis.									
++	++	++	++	++	++	...	±	±	—
++	++	++	++	++	++	++	±	—	—
++	++	+	±	—	—	—	...	...	...

In primary and secondary syphilis, then, not only are such lesions as can be observed rapidly removed by intensive treatment, but in addition the Wassermann reaction tends to change even in the early weeks from a positive to a negative state. This favourable change in the Wassermann reaction shows that treatment is bringing about a process of cure, but it is not sufficient to discontinue treatment at this stage, for unless treatment of a suitable character is maintained for a sufficient period a positive phase of the Wassermann reaction will usually reappear. From such uncured cases the various types of late syphilitic disease arise and in view of the serious nature of many of them it would seem completely justifiable in the early or curable stage to subject the patient to a prolonged and severe course of treatment. While syphilologists differ as to the exact manner in which they employ the available remedies most concur in the belief that two complete years should elapse from the time that infection occurred before treatment is discontinued. This plan has been in operation at the Middlesex Hospital since 1912 and the method has been found to give a high percentage of successful results. The details of the method of treatment employed at present are as follows: First, a course of ten intravenous injections of a "914" compound is given at weekly intervals, the first dose being 0.6 grm., the subsequent doses of 0.9 grm. each. This is followed by twelve intramuscular injections of mercury also at weekly intervals and then the Wassermann reaction is tested. If negative, mercurial treatment is continued until two years have been completed; if positive, the "914" course is repeated.

The object of this scheme is first to reduce the degree of infection by the salvarsan compound and then to complete the cure with mercury. In some cases actual cure may be brought about by the first series of injections, and this has been shown in certain cases where the patients have only attended for a few weeks, and where on their return, after a considerable interval during

which no treatment has been given, the disease is found to have been cured. But since it is impossible to tell in any given early case how much treatment will be required the pace should be set by the slowest and the prolonged course adopted. The dosage must of course be suitably modified where circumstances demand it.

When the response to treatment as shown by the Wassermann reaction in old-standing disease is examined completely different results are found. The following examples of such cases under intensive treatment with "914" are given.

LATE SYPHILIS: WEEKLY OBSERVATIONS OF THE WASSERMANN REACTION.

1	2	3	4	5	6	7	8	9	10 weeks
++	++	++	++	++	++	++	++	++	++ 18 years' duration
++	++	++	++	++	++	++	++	++	++ tabes

Thus, in late syphilis it would seem that the principles of treatment are bound up with the consideration of the clinical and serological reactions. If our object be to obtain cure of these cases—that is to remove the lesions and also to establish a permanently negative Wassermann reaction—then a prolonged and severe course of treatment would be justified. But the establishment of a permanently negative Wassermann reaction in such cases can but rarely be hoped for, and in consideration of this fact it would seem that the greatest benefit is to be obtained by short intensive courses of treatment which have as their object improvement of the symptoms by modifying or removing the lesions. Such treatment may consist of the ten intravenous injections of "914" already outlined, followed by twelve mercurial injections. Since, apart from a few exceptional cases, disease is not cured but only relieved by remedies so employed, it follows that a repetition of this course from time to time is indicated, this being regulated by the requirements of the individual case.

Between definite early syphilis and definite late syphilis there exists an intermediate class where cure may still be hoped for. This class includes patients who have received an insufficient treatment in the early stages, and in whose blood some two or three years later the Wassermann reaction is found to be positive. This group is important, because it includes certain cases in which the examination of the cerebro-spinal fluid reveals early involvement of the central nervous system. Here it would seem that a prolonged treatment is justified since there is still a possibility of cure.

The second point to be dealt with is the belief that neuro-syphilis is caused by a special form of spirochæte. Consideration must be given to this possibility, for if this belief be accepted it may be necessary to employ special remedies or remedies in a special manner. There is a certain amount of clinical evidence in favour of this view. Dermatologists have observed that where late lesions appear on the skin it is the rule that the nervous system escapes; the converse would also appear to hold good. But this rule is not absolute, and recently two exceptions to it have come under my observation: the first, the case of a man with syphilis of the nervous system and a late eruption on the legs, and the second the case of a woman who developed hypodermic gummata and neuro-syphilis. These two cases would appear to throw some doubt on the validity of a rule of very general application and suggest that the same spirochæte can attack both skin and nervous tissue. It should also be remembered in this connexion that the mucous membrane of the mouth is not infrequently involved in all forms of late syphilis, and that leukoplakia of the tongue

may be observed both where there is *tabes* and where there are cutaneous *gummata*.

Marie and Levaditi, in their well-known paper on this subject, recall the statement of Fournier that, in the future general paralytic, syphilis begins and evolves in a special manner: that the primary lesion may be of transient character, and that the secondary eruptions are ephemeral or non-existent. It is certainly true that many patients with neuro-syphilis deny all knowledge of an early infection, and this would seem to be in accord with Fournier's observations; but this negative history is equally common among those who present late cutaneous lesions. It would appear, in consideration of the above facts, that the existence of a neurotropic spirochæte is not established and therefore, so far as we know, there is no reason to modify treatment to meet the possibility of a special infection.

My next allusion is to the alterations in the cerebro-spinal fluid in the early stage of disease. Many observers have noted such alterations and some argue that they indicate a future involvement of the central nervous system. These changes include an increase of cells and of globulin and the presence of a positive Wassermann reaction. Weichselmann has recorded changes in the cerebro-spinal fluid in the pre-roseolar period, and Plaut has reported the finding of spirochætes in the cerebro-spinal fluid of patients in the early stage. Different figures are given by different observers as to the frequency of these early changes. Thus Ravaut computes them at 68 per cent. while Dreyfus gives so high a figure as 80 per cent. Although these findings may be regarded as only indicating the extension to the nervous system of a general syphilitic septicæmia they do in fact show that the central nervous system is actually involved. Now we know that the proportion of ultimate syphilitic disease of the nervous system is nothing like so high as these figures indicate. From this it may be reasoned—first, that syphilis of the central nervous system in its early stage is curable; and secondly, that remedies introduced into the blood in the ordinary course of treatment have a full curative effect on disease of the central nervous system. Here, however, it should be noted, we are dealing with the disease in an early stage where the general rule as to its curability holds good even for the central nervous system.

Reference may be made to the work of Fildes, Parnell and Maitland. They examined the cerebro-spinal fluid of a series of cases before and after routine treatment. Among the earlier cases, that is, among those with an infection of eighteen months' duration or less, out of a total of sixty-one cases, in thirty-three the Wassermann reaction in the cerebro-spinal fluid remained negative before and after treatment; in the other twenty-eight cases, in eight the Wassermann reaction in the cerebro-spinal fluid changed from positive before to negative after treatment, in fifteen it was found positive both before and after treatment and in five cases it altered from negative before to positive after treatment. These results may not appear to be very good but it should be pointed out that the total treatment was limited to some 2 or 3 grm. of "914," an amount that will be admitted to be very small.

When we contemplate late stages of syphilitic infection of the central nervous system we find exactly the same difficulty in favourably influencing the Wassermann reaction as in late syphilis generally. It may be stated broadly that two types of lesion exist, those of the parenchymatous tissues and those of the interstitial tissues. Little benefit can be expected in parenchymatous disease and therefore our methods should aim at relieving interstitial disease. The question of treatment is complicated by the physiological



peculiarities of the cerebro-spinal fluid and the difficulties of affecting it by intravenous medication. Nevertheless we have evidence of the effect of intravenous medication in neuro-syphilis in the early stages of disease and there is no reason to believe that in the later stages the interstitial tissue may not receive benefit from intensive intravenous medication. This beneficial effect is found to occur in another type of syphilitic disease where it might be expected that but little or no improvement would result from the injection of salvarsan into the veins—namely, syphilitic interstitial keratitis. Here a tissue completely free from blood-vessels, except when acutely inflamed, may be profoundly influenced through the general blood stream. If antisubstances produced by intravenous medication can reach such a tissue through the blood it is not difficult to believe that the central nervous system can also be profoundly influenced through the general blood stream.

Lastly, to deal with the general question of the special treatment of neuro-syphilis. Many believe that spinal drainage increases the effect of intravenous treatment. The secreting mechanism is rendered more permeable, enabling substances to pass from the blood into the cerebro-spinal fluid. Where the intention is to carry "antisubstances" into the cerebro-spinal fluid this procedure would appear to be logical, but if the intention is to introduce arsenic then the operation is open to criticism. When another method of treatment is considered, namely, the introduction of salvarsanized serum into the cerebro-spinal fluid, the same criticism may be applied, for it has been shown that the "606" and "914" compounds have no direct action on the *Spirochaeta pallida*, but that they act through the tissues indirectly by causing them to elaborate the "antisubstance." It is not clear how these antisubstances can be produced by the introduction of minute quantities of arsenical substance into the cerebro-spinal fluid, a situation not particularly favourable to their elaboration. Nor is this procedure comparable with intrathecal injections in tetanus or cerebro-spinal meningitis, for there an antitoxin is given to neutralize a toxin. It may be argued that the serum taken from patients by the Swift-Ellis method contains a potent antisubstance. On this point I can bring forward some evidence. At one time I injected the serum taken from patients with secondary syphilis undergoing intensive arsenical treatment into certain untreated patients, and in no case could I observe any effect on the disease by these methods. As such serum presumably containing antibodies had little or no effect on secondary cutaneous eruptions it is unlikely to benefit the lesions of neuro-syphilis when introduced into the cerebro-spinal fluid.

Those who advocate intraspinal medication have recorded benefits where intensive treatment has been previously carried out without apparent benefit. In these cases the intrathecal treatment is frequently undertaken after a preliminary intravenous treatment. Through the kindness of my colleague, Dr. Campbell Thomson I have had the opportunity of treating a series of cases of neuro-syphilis. I have used the intravenous method and I have observed that when improvement occurs it is usually delayed for two or three months, and it is not improbable, in view of this observation, that in many of the cases where benefit is attributed to intrathecal medication it should rather be attributed to the previous intravenous treatment, the improvement corresponding with the period at which intrathecal injections were commenced.



## Sir JAMES PURVES-STEWART, K.C.M.G., C.B.

said that last summer, in Glasgow, he had opened a Discussion on the Treatment of Neuro-syphilis, and his paper had appeared, *in extenso*, in the *British Medical Journal*, of October 7, 1922. He did not propose to re-state the main points of that contribution, more especially as Dr. Buzzard had alluded to some of those points in his opening address. He felt a little disappointed that Dr. Buzzard did not sufficiently come down to the hard facts of clinical treatment. He was still eager to know what was Dr. Buzzard's usual treatment of neuro-syphilis.

He (Sir James Purves-Stewart) did not think there was any routine or stereotyped treatment for neuro-syphilis at the present time. Every case of the condition demanded assiduous and life-long treatment of the general infection by every means at disposal. In no case should remedies be directed exclusively to the nervous system.

He had hoped that the various methods of treatment would be discussed on the present occasion, e.g., by mercury, salvarsan, iodide of potassium, bismuth, malarial infection, pyrexial treatment by nucleinate of soda, spinal drainage, &c., and the various special indications for each. The choice between these must not be regarded as a contest between rival methods of treatment; there should be no rival methods, but each should supplement the other. It would be agreed that some physicians acquired special experience by employing one line of treatment more than another, and in that way they might become biased in favour of some special form.

He had been asked to deal, in this discussion, with the intracisternal treatment of selected cases of general paralysis. It consisted of the injection of salvarsanized serum into the cisterna magna, the beneficial action being probably due not to the infinitesimal amount of salvarsan the injected substance contained, but to the antibodies evoked in the serum of the patient.

A man, aged 43, who came from South America, acquired syphilis many years ago, and he had had no treatment for it. He had Jacksonian fits affecting the right side of the face and the right arm, followed by temporary dysarthria. The only physical signs discoverable were an extensor plantar reflex on the contralateral side, and absence of one abdominal reflex. He evidently had multiple lesions. The cerebro-spinal fluid showed 142 cells per cubic millimetre and the Wassermann reaction was strongly positive. The intravenous introduction of salvarsan was at once begun, and on the same day withdrawal of blood was carried out, and on the day following salvarsanized serum was injected into the cisterna magna. The improvement was dramatic in its rapidity (see table). When next the cerebro-spinal fluid was withdrawn there was a

Date	Blood Wassermann	Treatment intravenous N.A.B.	Cerebro-spinal fluid		Treatment, intracisternal salvarsanized serum
			Cells	Wassermann	
1921. Nov. 3	...	...	142-19	+++	...
Nov. 15-16	... + ± -	...	81.4	++	...
Nov. 29-30	... + ± -	...	68.4	+	35 c.cm.
Dec. 13-14	... + ± -	...	62.5	-	...
Dec. 27-28	... + ± -	...	27.08	-	...
1922. Jan. 10-11	... + - -	...	9.7	-	...
May 2	...	...	6.3	-	...
Nov. 17	...	...	...	...	...
1923. May 20	...	...	1.8	-	...

fall in the cell-content to 81, and progressive withdrawals and examinations gave 27, 9.7 and 6.3. That progressive difference, from 81 to 6.3 cells per cubic millimetre,

took place in the few weeks between November and January after five treatments. The blood Wassermann test, at first partially positive, became almost completely negative, and then quite negative. The cerebro-spinal fluid was strongly positive at first, but soon was negative, and remained so. When he last saw the man, two and a half years afterwards, he was free from physical signs. The fits had stopped soon after the treatment was commenced, and his reflexes had since remained normal.

The table exhibited gave various details of a series of cases of neuro-syphilis (see p. 71).

A man, aged 63, a Naval officer, acquired syphilis rather late in life, and developed intense emotional attacks, almost amounting to acute melancholia. The only objective clinical sign he (the speaker) could ascertain was slight rigidity at the back of the neck. The cerebro-spinal fluid, however, contained 163 cells per cubic millimetre, and the Wassermann reaction was positive for both blood and cerebro-spinal fluid. Salvarsanized serum was injected into the cisterna magna, and after this injection the blood Wassermann was feebly positive. The patient now had a completely negative reaction both in the blood and cerebro-spinal fluid, and the cells in the fluid had fallen to normal. The mental symptoms had entirely cleared up as well.

The course of the other cases was less dramatic. One was that of a man, aged 42, whom he showed to Professor Babinski last July. He had unilateral Argyll-Robertson pupil, some weakness of the right hand, and extensor plantar reflexes; obviously he had multiple syphilitic lesions. Under intra-cisternal treatment the cell-content fell from 189 to 9 cells per cubic millimetre; but the Wassermann reaction both in blood and cerebro-spinal fluid remained positive. He was not regarded as cured and treatment was still being persevered with.

There was also a case of a patient with early general paralysis, who began with 44 cells, and they were reduced to 2 per cubic millimetre. But in his case also the Wassermann still remained positive for blood and cerebro-spinal fluid.

General paralytics improved under the treatment to some extent, but they did not get back to the normal as regards the Wassermann reaction. He had never yet seen a general paralytic in whom the Wassermann reaction had been rendered negative in the cerebro-spinal fluid, though he had seen many instances of that disease in which the cell-content had fallen to normal.

In order to illustrate the method Sir James Purves-Stewart demonstrated the anatomical relations of the skull. The substance injected into the cisterna magna circulated slowly; some of it passed down the spinal canal, but most of it circulated forward along the basilar cistern, into the inter-peduncular cistern; from this it circulated upwards above the corpus callosum, some up the Sylvian fissure, and ultimately it bathed the whole cortex, becoming ultimately re-absorbed through the capillaries of the cortex. Sir Charles Ballance and Dr. Harry Campbell, some years ago, in conjunction with himself (Sir James), had tried the effect of doing callosal puncture, and introducing salvarsanized serum into the lateral ventricles; but it seemed a severe method of treating this disease (general paralysis of the insane), whereas treatment by the injection of the cisterna magna could be done much more easily and without causing surgical shock.

With regard to the technique of cisternal puncture, the skull was entered through the atlanto-occipital ligament, which brought the operator to the cisterna magna. The floor of the fourth ventricle was in front. There was a space extending to a depth of  $\frac{1}{4}$  to  $\frac{3}{4}$  in., a space of safety within the cistern. The needle was directed at the proper angle through the atlanto-occipital

INTRACISTERNAL SERUM TREATMENT OF NEURO-SYPHILIS AND GENERAL PARALYSIS OF THE INSANE.

No.	Initials	Date	Age	BEFORE TREATMENT			No. of injections	AFTER TREATMENT			Clinical phenomena	Results
				Blood	Cerebro-spinal fluid	Wasser-mann reaction		Blood	Cerebro-spinal fluid	Wasser-mann reaction		
1	W. L. W.	November, 1920	43	+ ± —	142·2	+++	5	— — —	1·8	—	Jacksonian fits	Symptoms entirely disappeared; remained well 2½ years later
2	H. L.	October, 1920	30	+	44·0	+++	4	+	2·6	+++	Early general paralysis of the insane; facial tremors	Tremors less
3	C. R.	June, 1921	53	—	13·3	+++	3	—	9·3	+++	Right hemiplegia, vertigo, fits	<i>In statu quo</i>
4	W. B. W.	July, 1921	40	+++	74·7	+++	4	+ ± —	5·6	+++	Emotional attacks; early G.P.I.	Mental condition improved
		Re-examined June, 1922			25·6	+++					Slight mental deterioration	Resuming treatment
5	H. B.	May, 1922	42	+	189·4	+++	4	+	9·0	+++	Unilateral Argyll Robertson pupil; weakness of right hand; extensor plantar reflexes	Rapid improvement in hand weakness
6	F. M. B.	June, 1922	63	+++	163·9	+++	4	+ ± —	7·4	—	Emotional attacks; no physical signs, apart from blood and cerebro-spinal fluid	Rapid disappearance of all symptoms

ligament. The salvarsanized serum was rapidly absorbed. To suggest this operation to a surgeon afforded a good opportunity of diagnosing the surgical mind by a psycho-motor test. It was almost certain that the surgeon, when this method was explained, would say it was an "anatomical impossibility." It really meant the surgeon thought the operation could not be carried out with safety. The requirements for it were three in number, viz.: a knowledge of anatomy, a steady hand, and a stout heart.

Colonel L. W. HARRISON, D.S.O., R.A.M.C.,

said that he believed in continuing treatment of syphilis for considerably longer than was originally thought necessary. The longer he lived, the more convinced he was that the production of a negative Wassermann reaction by no means meant the end of the syphilis. One could influence even general paralysis by antisyphilitic treatment, provided it was sufficiently intense. He thought the number of cases of this disease might be considerably reduced if the patient in every case of syphilis underwent an examination of the cerebro-spinal fluid some years after the original infection; he agreed with Ravaut that five years after infection would be a good time. That authority said if the fluid was found negative then, he had not found it become positive subsequently. There might be exceptions to that, but it seemed a good rule.

Dr. MacCormac had voiced the view that a positive Wassermann reaction indicated activity of the spirochæte; with that he (Colonel Harrison) agreed, though other views were held. He preferred to look for the reaction in both the fluid and the blood.

The question of the possible duality of *Spirochæta pallida* had also been raised. He found difficulty in accepting this theory. If there were a special neurotropic virus, how did it get into other people? And how did it explain the frequency of aneurysm and cardiovascular lesions in tabes and general paralysis?

With regard to the administration of arsenobenzol in cardiovascular cases he was interested recently, in a case of general paralysis of the insane, to see the large amount of arsenobenzol a patient with cardiovascular lesions could take, and he thought there was need for a revision of views on that question. Though this patient had a double aortic murmur he (the speaker) had repeatedly given him a gramme of silver salvarsan in a single dose and it did not particularly upset him.

Dr. MacCormac had mentioned that in early cases he treated intensively with arsenobenzol, and then continued for two years with mercury. He (Colonel Harrison) had never been able to understand why the continuation treatment should not be with arsenobenzol. Presumably the continuance on mercury was based on the assumption that the spirochæte was still alive. If it was—and however latent it might be, it might beat the patient in the end—why not hammer the spirochæte with all the remedies at our disposal?

With regard to intrathecal therapy, he had seen some apparently good results from this when the intravenous method had not seemed to lead to much progress. He was, however, rather sceptical as to the effect being due to the specific action of the remedy introduced intrathecally. He had seen symptoms alleviated by mere lumbar puncture, and he had never been able to convince himself whether with intrathecal injections this was due to the specific action of the remedy on the parenchyma, or simply to the disturbance that took place as a result of the lumbar puncture. He remembered two

cases in which there was a very definite effect from lumbar puncture without anything else being done. Both were cases of tabes with paralysis of the bladder, and in both the control of the bladder had returned twelve hours after the puncture.

Dr. HENRY HEAD, F.R.S.,

said that Colonel Harrison spoke of general paralysis as if it were a disease which could be diagnosed with certainty from other conditions due to syphilis. General paralysis was only a name for a group of signs and symptoms. When one took a number of cases having that group of signs and symptoms, the dementia, the pupil phenomena, tremor of hands, &c., and tested the blood and cerebro-spinal fluid, one found that the cell-content was increased, and that both the blood-serum and the cerebro-spinal fluid gave a positive Wassermann reaction. If during active treatment watch was kept on the cerebro-spinal fluid and on the blood, one found that those patients were divisible into two groups. In one of the groups the cells diminished materially, the Wassermann reaction both in blood and cerebro-spinal fluid improved, and might become negative in six months. This was an example at one extreme pole.

On the other side, one found that at the end of six months of treatment the Wassermann reaction remained the same as before; the cells might have decreased somewhat, but the patient's symptoms were not materially improved, and there was nothing to indicate that any effect had been produced on the disease.

Between these extremes there were a complete series of intervening links. The condition of some patients seemed to have been improved a little, some still more, up to the degree where the cells in the fluid were reduced to normal and the Wassermann reaction became negative.

When an observer said that general paralysis was not influenced by treatment, it meant that a certain group of signs and symptoms had not been affected. But if he had taken a series of cases which presented all the signs of what was known as general paralysis he would have found that treatment produced the most different results. One of the patients in his (Dr. Head's) series was in an asylum and was diagnosed as a straightforward case of general paralysis. The Wassermann reaction in his cerebro-spinal fluid became negative and quite recently he was known to be still alive and well. No one could say beforehand, except in the very few fulminating cases, whether an infection of cerebro-spinal fluid could be influenced by treatment, or which patients could be so influenced.

Sir James Purves-Stewart had said that among his patients who had undergone the cisterna magna treatment there was a group of general paralytics in whom the state of the Wassermann was not influenced by the treatment, and the patients did not appear to be improved. That confirmed the experience of Dr. Fildes, Dr. Mackintosh and himself (cf. *Brain*, 1916, xxxix, pp. 478-83).

When patients were injected with a mercurialized serum he understood it was a serum to which some perchloride of mercury was added. This was said to produce a tremendous reaction, a rise of temperature, with increased symptoms, followed, often, by improvement. What he desired to know from those who had used this method was: Had the mercury anything to do with it? Was not the mercury made innocuous by forming an albuminoid in the serum? And was not the reaction really due to protein shock?

## Dr. G. RIDDOCH

said he thought one point had not been sufficiently emphasized in the discussion, namely, that the aim of treatment was, primarily, the eradication of the syphilitic infection. This could not always be achieved, but it must be attempted. It did not necessarily mean disappearance of the symptoms; the spirochæte might still be active although symptoms had gone, and too often treatment was stopped at this stage. Whatever view might be taken of the significance of the Wassermann reaction—it was sometimes found to be positive in some other diseases, such as scarlet fever and leprosy, and was said to be positive in pregnant women—still it was almost always positive at certain stages of syphilitic disease after the primary inoculation of the infection, and in neuro-syphilis, except during quiescent phases in tabes, &c. Therefore it must be accepted as indicating activity of the syphilitic process. Of this there were other indications as well, such as lymphocytosis in the cerebro-spinal fluid, which should be used in conjunction with the Wassermann test. He would emphasize that treatment should be continued not only until symptoms had disappeared or diminished as far as possible, but also at least until these tests had become negative or improvement in them had definitely stopped. The various tests should be used not only for diagnostic purposes but also as indications of the efficacy of treatment. The laboratory tests could not of course be separated from the clinical indications; the two should be used in conjunction.

## Dr. C. P. SYMONDS

spoke of the possible occurrence of toxic phenomena as a result of dosages such as had been mentioned in this discussion. Eighteen months ago it seemed to be agreed that such dosages as Dr. MacCormac now advocated were dangerous, because of the risk of such sequelæ as toxic jaundice and exfoliative dermatitis. He (the speaker) had found that patients with neuro-syphilis had, on the whole, tolerated large and protracted doses of the arsenical compounds better than did sufferers from the primary disease. In the neurological department to which he was attached a beginning was made by the injection of 0.6 gm. of novarseno-billon, then by proceeding to 0.9 gm. and continuing until the patient had taken 10 gm. as a first course. But there were too many cases of jaundice and exfoliative dermatitis; therefore they now divided the course into two, with six weeks' interval between. He asked what was the experience of Dr. MacCormac in the matter of toxic effects.

With regard to the Wassermann reaction in neuro-syphilis, he agreed with what Dr. Buzzard said; one might find actively progressing tabes with a negative Wassermann reaction in both blood and cerebro-spinal fluid. With Dr. Osman, at Guy's, he had tried to measure the effect of treatment in a number of cases of tabes. It was very difficult, because of the spontaneous remissions and relapses; still, they hoped eventually to present some results. They had satisfied themselves that in certain cases of clinical tabes there might be active progress of the process while the Wassermann reaction remained negative for both fluids. One man had clinical signs of tabes, actively progressing, but his Wassermann was negative, both in blood and cerebro-spinal fluid. His wife however was persuaded to come to the hospital,



and her Wassermann reaction proved to be strongly positive. Even if, following treatment, a negative Wassermann was produced, that did not indicate that the disease had been cured.

Dr. A. FEILING

said the significance of the Wassermann test, in its bearing on diagnosis and treatment, was very important, because not only the profession, but the public, laid much stress on the state of the Wassermann reaction in the blood. He agreed strongly with Dr. Buzzard and Dr. Symonds in this respect, and was surprised to hear Dr. MacCormac say he regarded disappearance of the positive Wassermann reaction in the blood as synonymous with cure. A negative Wassermann reaction in the blood was often found in syphilis of the central nervous system, and it was not uncommon to find cases of undoubted syphilis of the central nervous system in which the patient's cerebro-spinal fluid also was absolutely normal. Recently he had had two such cases under his care. One was that of a woman whom he first saw early in 1915, when she was suffering from spastic paraplegia. The Wassermann reaction was then positive, in both blood and cerebro-spinal fluid, and the case was regarded as one of chronic syphilitic meningo-myelitis. She had had a variety of anti-syphilitic treatment over a long time, and now she was attending the outpatient department, and was taking mercury. But her symptoms had distinctly progressed, and she had now marked paraplegia and gross loss of sensation, some muscular wasting of the arm and diminished arm reflexes; the cerebro-spinal fluid was now normal.

The other case was that of an elderly gentleman who had the classical signs of tabes. His blood Wassermann was negative, and cerebro-spinal fluid normal, yet his lightning pains were so severe that he had to be given morphia occasionally.

He would dread giving a case of neuro-syphilis the repeated large doses of arsenical compounds that had been mentioned that evening; he had seen severe damage to the nervous system result under such conditions. Eighteen months ago a man came under his observation with the history that he went to a hospital on account of a trivial complaint unconnected with syphilis. He told the doctor he saw, that he had had syphilis and had been treated for it. The doctor then advised that the blood should be examined. It was found to be positive; he was given nine intravenous injections of an arsenical compound at weekly intervals. Six weeks after the last one he rapidly became completely paraplegic with acute myelitis, from which he was now very slowly emerging.

A boy who was in the Navy during the war was discovered, at the age of 17, to have secondary syphilis and a positive Wassermann reaction. Within ten days he was given three intravenous injections of N.A.B., each of 0.45 grm. After the third dose he was comatose, paralysed, and incontinent. He also was now making a slow and painful recovery. He had wondered whether the patient to whom Colonel Harrison gave such large doses of silver salvarsan had yet had time to exhibit the serious toxic effects which might ensue.



## DR. WILFRED HARRIS (President)

said he thought the note of warning sounded by Dr. Symonds and Dr. Feiling was very necessary.

His own experience was very similar to that of Dr. Buzzard, that the Wassermann reaction in neuro-syphilis was of comparatively little value. A case should be treated more on the clinical signs than on any biological reactions. If a positive or negative reaction was to be set up to be shot at, then in the process of treatment so based the patient might be killed. He had himself seen most acute nephritis result from arsenical treatment in the V.D. clinic several times; he had had to treat the cases in the wards afterwards. He was sure weekly-repeated doses of N.A.B. constituted a grave danger in many cases.

He would like some attention to have been given to the comparative value of arsenical preparations. His own experience was very much in favour of the old salvarsan, or the substitute kharsivan. He had seen cases which had been extensively treated by N.A.B. and galyl without benefit, clear up almost magically under intravenous injections of salvarsan.

An Australian officer acquired syphilis in the war, and had the disease in the cerebrium three months later; there were partial hemianopia, headache and fits. He was treated at a military hospital by an expert from the Lock Hospital with galyl, of which he had several injections. But the fits and hemianopia remained, and he appeared to be in a bad way. Against military etiquette, he persuaded the officer to enter a nursing home and receive salvarsan injections at his (the speaker's) expense. Two injections of this made an enormous difference; the hemianopia disappeared, and he had no more fits.

Another patient had had syphilis eighteen months before, and was treated by nine doses of neo-salvarsan by an expert engaged in a venereal clinic. This man's severe headaches became no better under this treatment. His doctor regarded the condition as functional, and told him to get out. The man complained to his doctor in Brighton, and he (the President) saw the patient and did a lumbar puncture. He had never seen so many cells in a clear cerebro-spinal fluid as in that case. He gave the man three intravenous doses of salvarsan, and gave intrathecal treatment, but he thought it was the salvarsan which did the good. The headache improved, the cells diminished in number, and he was well of that part of his trouble. But he (the President) believed the amount of three doses was too much; he now only gave two of 0.6 gm.; he had seen bad results follow the third injection, such as tinnitus, and from a third dose of 0.6 gr. of kharsivan he had seen in tabes what he called "wooden leg," that was, the man complaining that his leg felt numb and heavy. These cases showed the need of caution in administration.

He had had great experience in the intrathecal treatment, perhaps on slightly different lines. He always waited a fortnight after an intravenous injection of salvarsan before drawing the man's blood, and giving serum on the following day. There was then no question of salvarsanized serum. What happened in tabes was nearly always striking. If a man with tabes had been suffering severe pains beforehand, the intrathecal injection brought on very severe pain in one and a half to four hours, so severe as to require morphia for twelve to eighteen hours, and then, in very many of the cases, the pains disappeared entirely, and they might not return for a year or longer. He had seen some cases in which there had been no return of pain, and the man seemed much better. Therefore there was a distinct field for intrathecal injection. In cases exhibiting the group of clinical phenomena called G.P.I.,

he had seen very little good result with intrathecal autoserum, but he was carrying out the pyrexial treatment in three cases. But he did not feel himself bold enough to hire a patient with active malaria and inject a syphilitic patient with that disease. He had been using anticitarrhal vaccines, and one case of G.P.I. he had been treating in that way had become extraordinarily better, though intravenous salvarsan and intrathecal serum had been employed without much benefit. The other two were also distinctly improved mentally.

DR. E. FARQUHAR BUZZARD

(in reply) pointed out that as Dr. MacCormac had based his observations and conclusions entirely on the Wassermann reaction, he could not agree with the conclusions at which he had arrived.

Sir James Purves-Stewart had blamed him (the speaker) for avoiding hard facts with regard to the routine treatment of neuro-syphilis; yet, in the same breath, he pointed out that it was impossible to lay down a routine treatment and that each patient had to be treated as an individual; and there he agreed with Sir James.

With regard to Sir James Purves-Stewart's work on cisternal injections, the cases he had brought forward did not convince him (the speaker) that there was any advantage in the method. The cases to which Sir James referred as satisfactory were of the kind in which one would expect satisfactory results from intravenous or intramuscular injections, and he did not understand why the satisfactory result was attributed to injecting salvarsanized serum into the cisterna when the patient had already had salvarsan injected into the vein. Moreover, Sir James had rightly said it was an operation that required a stout heart—on the part of the patient as well as of the operator.

DR. H. MACCORMAC, C.B.E.

(in reply) said that as regards the toxic effects to which Dr. Symonds had referred, he could only state that in his personal experience such were not common; arsenical dermatitis and jaundice did occur from time to time but they were not confined to cases in which heavy dosage had been given. He ought, perhaps, to have made it clear in his remarks that he referred to the treatment of late syphilis in general—not neuro-syphilis in particular. Where the condition of the patient demanded it the dosage was suitably modified. Colonel Harrison asked why more than one series of salvarsan injections should not be given. Where the Wassermann reaction was found positive in early cases after a first course of arsenic and mercury a further series of arsenical injections should be given. Mercury was, however, a much safer remedy than salvarsan and if after the preliminary series of arsenical injections cure could be effected by mercury, then it would appear undesirable to subject the patient to the risk incident to further arsenical administration. This form of treatment—a preliminary course of salvarsan followed by two years' mercurial administration—had been carried out in the Middlesex Hospital Clinic since 1912 and the results had been most satisfactory. The President had spoken of the undue reliance placed upon the Wassermann reaction in the older stages of syphilis. He (the speaker) would point out that little or no

attention was paid to the Wassermann reaction in such cases for reasons already given. Dr. Feiling raised the question of the disappearance of the Wassermann reaction as a sign of cure. A temporary alteration from positive to negative might mean little or nothing, but where the negative reaction in the blood and cerebro-spinal fluid was maintained over a number of years there would appear to be good reason to believe that cure had been established.

## Section of Neurology.<sup>1</sup>

President—Dr. WILFRED HARRIS.

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### Case of Progressive Double Athetosis.

By A. FEILING, M.D.

PATIENT, a boy, now aged 9, was normal at birth and continued so until August, 1919, when, as the result of a street accident, his right foot was amputated. After recovery, and when he began to use crutches, involuntary movements were noticed in both arms. Later these movements affected the legs as well. Three years ago dribbling from the mouth began, shortly followed by a progressive loss of speech. Until about eighteen months ago he was able to swallow well but during the last year spasmodic movements of the jaws have seriously interfered with feeding. The mental faculties have not suffered.

Past history: Measles, chicken-pox and erysipelas.

Family history: No history of any nervous or mental disease. Three other children alive and well; one child died of pneumonia; one miscarriage three months after marriage.

On examination he is seen to be undersized. He is now quite unable to speak but he can indicate by signs what he means and is perfectly intelligent, though the mental state is retarded for his years.

Pupils and optic discs normal. No paralysis of ocular muscles or of face. Constant spasm of jaws leading to marked grinding of the teeth and interfering with feeding and mastication. The tongue now can hardly be protruded at all and, when it is, is deviated to the left. Swallowing very difficult and food sticks in the throat.

The arms, the left especially, are the seat of involuntary movements. These take place at all the joints but are most marked at the shoulders. They more resemble the type of athetosis than anything else. No actual paralysis. Not much alteration in muscular tone but some hypertonus is perhaps present. Arm-jerks present. Similar movements affect the legs but the right is the more affected.

The knee-jerks and the left ankle-jerk active. Plantar response somewhat doubtful, probably flexor. No loss of sensation has been found. He is quite unable to stand, even with assistance and with a peg leg, but can sit up unaided.

One year ago the Wassermann reaction in the blood was feebly positive. The cerebro-spinal fluid was quite normal. The Wassermann reaction in the blood of the mother is negative. He was treated a year ago with antisyphilitic remedies, mercury and iodide and intramuscular injections of glucarsenol. Some improvement took place but recently there has been a slow deterioration, especially in the power of swallowing.

<sup>1</sup> Cases shown at the Clinical Meeting held at the Royal Society of Medicine, 1, Wimpole Street, W., on Thursday, May 10.

### Case of Chronic Mercurial Poisoning.

By E. A. CARMICHAEL, M.B.

PATIENT, a male, A. S., aged 51, thermometer maker.

History: Eighteen months ago teeth became black and loose. Twelve months ago gums were painful. Three months ago salivation and unsteadiness of hands with "trembling" of legs. Two months ago difficulty in going upstairs, followed later by weakness in wrists. Seven weeks ago bad occipital headaches and aching in knees and wrist. Looseness of bowels of indefinite duration. During the last three months memory very bad and speech slurred. No history of stabbing pains, vomiting, delusions or obnoxious taste.

On admission: Skin of body and especially face dirty sallow colour. Teeth few in number, black, loose with retracted gums. Tongue raw looking. Arteries slightly thickened. Fundi normal. Pupils equal, no ocular signs. Lower jaw trembling as if silently mumbling. Hands and legs showed irregular tremor. Grasp poor, extensors of wrist weak. Plantar flexion of both ankles weak. Reflexes normal. Diminution of appreciation of pinprick and cotton wool of glove distribution on arms and also over legs and lower trunk. Blood count and picture normal. Gastric contents showed diminution of free HCl. Cerebro-spinal fluid and blood normal.

The patient has improved considerably since admission.

### Case of Muscular Atrophy of the "Peroneal" Type apparently commencing in, and confined for some time to, the Right Hand.

By C. P. SYMONDS, M.D.

THE patient, Mrs. C., aged 32, is one of a family in which I have found nine members affected by the disease in two generations. She belongs to the senior generation, and her father is also stated to have suffered from the disease. Her sister, Mrs. B., aged 38; her niece, Miss B., aged 15; and her nephew, Mr. William C., aged 17, are presented as typical cases of peroneal muscular atrophy.

The present patient, Mrs. C., states that she has noticed from childhood that the fourth and fifth fingers of her right hand would not straighten out properly. She has never been seriously inconvenienced by weakness of either hand. She was at school till the age of 14, then in domestic service for six years, married at 23, and has two children aged 8 years and 2 months, whom I have seen, and who appear normal in every respect. The deformity of the right hand was recognized by the family from childhood. She states that she has not suffered from any illnesses except occasional headaches, and that she has not noticed anything wrong with her feet.

Her general condition is good, and her mentality normal. The right hand shows wasting of the intrinsic muscles of moderate degree. This is essentially of the ulnar type. The intrinsic muscles of the thumb are slightly, if at all, affected. The hand is deeply hollowed, the hypothenar eminence is lacking, all the palmar and dorsal interossei are wasted and weak, the wasting and weakness roughly corresponding. The left hand is relatively normal. Both

feet show pes cavus with claw toes. This is of moderate degree and more pronounced on the right than on the left. Power in the feet and legs is good on the whole though dorsiflexion is somewhat weak on the right. When examined a month ago fibrillary twitching was noted in the peronei of both legs, being more noticeable on the right. Sensation to cotton-wool and pin-prick is normal, but sense of position is slightly impaired in the toes. Sensibility to the vibrating tuning-fork estimated quantitatively appears diminished both in legs and arms. The ankle-jerks could not be obtained; knee-jerks sluggish, obtained with reinforcement; triceps-jerks, left greater than right; supinator-jerks, not obtained; abdominals present and equal; plantars indefinite.

#### DISCUSSION.

Dr. STANLEY BARNES referred to a series of cases, which he had found, of a somewhat similar type of myopathy. In this series a strong inheritance had been observed and the cases had been traced back as far as 1804. One of the most unusual features was the degree of variation at the different periods of life, so that some of the cases appeared at one stage to be of the true hypertrophic type, at another of the juvenile type and in one case at a late stage it might have been thought that the patient was suffering from the distal type of myopathy.

Dr. F. J. NATTRASS said that he had recently seen two cases of this disease in brothers, who had double drop-foot due to wasting and weakness of the anterior tibial groups of muscles: in each patient both peroneal groups still retained good voluntary power. Strong faradism failed to elicit any response in the tibiales antici muscles in these cases, but the response to galvanism, though weakened, was almost as brisk as normal. The peronei responded well to faradism.

### Case of "Juvenile" Tabo-paresis.

By C. WORSTER-DROUGHT, M.D.

R. P. B., MALE, aged 26. No history of any illness of importance before onset of present disorder. Was up to the average standard at school; on leaving, worked as a "light porter" in Government service until his mobilization with Territorial Forces in August, 1914. In January, 1917, he appears to have had a "seizure," and, as a result, was discharged from the Army in May, 1917. He returned to his civil employment and continued in it until 1919, when increasing mental deterioration and the fact that "he was always falling asleep" led to his dismissal.

Present condition: Invariably cheerful and self-satisfied; childish and inane, but usually answers questions fairly rationally; inclined to be grandiose in his ideas, with some degree of confusion: memory for past events very poor and orientation fairly good. No delusions. Pupils: Left larger than right, irregular in outline with no reaction to light direct or consensual. Optic discs somewhat pale. Hutchinsonian teeth. All arm-jerks brisk, knee- and ankle-jerks absent. Ulnar nerve and tendo-Achillis analgesia. Frequent urinary incontinence. Some unsteadiness of gait, but Rombergism not marked. Blood: Wassermann reaction positive. Cerebro-spinal fluid (last examination) 272 lymphocytes per cubic millimetre, globulin and Wassermann reactions positive, colloidal gold curve of "paretic" type.

Under observation and treatment with novarsenobillon intravenously and

## 82 Worster-Drought: *Juvenile General Paralysis; Chorea*

mercury cream intramuscularly at intervals during the past four years, patient has shown very little change, excepting for a steady increase in the cell content of the cerebro-spinal fluid from 88 cells to 272 cells per cubic millimetre.

### Case of Juvenile General Paralysis of the Insane.

By C. WORSTER-DROUGHT, M.D.

C. M. D., MALE, aged 22. Patient left school in Standard VI at the age of 13, having had no illnesses except attacks of bronchial catarrh each winter. When he was 14 years old he began to "stammer," but was able to carry on with his work at Woolwich Arsenal until he was 18 (four years ago); he then had a "fit," and was unconscious for twelve hours. Since that time he has steadily deteriorated, both mentally and physically. His father died sixteen years ago (aged 40) from pneumonia, while his only brother, aged 15, has interstitial keratitis. His mother is alive and well.

Present condition: Mentally dull and stupid, and easily amused, but occasionally he answers questions fairly intelligently. Pupils: Right greater than left, inactive to light direct and consensual; optic discs—physiological cups, but not unduly pale. Arm-jerks brisk and equal, abdominals brisk and equal, knee-jerks both present, ankle-jerks unobtainable, plantars flexor. No ulnar nor tendo-Achillis analgesia. Gait uncertain and unsteady but no Rombergism. Speech slurred. Blood: Wassermann reaction positive. Cerebro-spinal fluid (last examination) 82 lymphocytes per cubic millimetre. Globulin and Wassermann reactions positive.

During the past three years he has been treated with courses of novarsenobillon intravenously and mercury cream intramuscularly; the only changes shown are: (1) Increasing tendency towards dementia; (2) loss of ankle-jerks, which may be a result of novarsenobillon.

### Case of Huntington's Chorea.

By C. WORSTER-DROUGHT, M.D.

J. T., MALE, aged 45, complains that he "cannot keep still," the continual movements resulting in insomnia. The condition developed in 1917 while the patient was serving in France but he was able to continue in the Army until 1919. In spite of a variety of forms of treatment the movements persist unchanged.

Present condition: If observed, he is seen, from time to time, to exhibit jerky movements of the head and limbs; the neck will be jerked backwards, the back straightened and the head then fall forwards, and the back become bowed. The hands may be thrown to one side with the whole arm sweeping outwards; while sitting he has found that his feet are continually shuffling owing to the leg movements. All the movements are loose, jerky, and show a wide excursion. It is understood that his wife is "always grumbling because he moves about so much in his sleep." Under hypnosis the movements continue but are less pronounced. Mentally, he is irritable, depressed, and his memory for past events very poor. Speech is indistinct, and somewhat grunting in character and is often accompanied by lateral movements of the head. The



pupils, fundi and other cranial nerves show no abnormality, the knee- and ankle-jerks are active and the plantar reflexes flexor. Blood: Wassermann reaction negative. Cerebro-spinal fluid: two cells per cubic millimetre; Wassermann negative; globulin slight increase; colloidal gold reaction, "luteic" curve (0.1.1.1.1.2.2.1.1.0).

Family history: Nothing of importance can be traced. He has been married six years and has no children. His mother died when he was 14 (thirty years ago)—of what disease he does not know—and his father as the result of an accident twenty years ago. He has a brother three years older than himself and a sister three years younger, both alive and well.

### Case of Quadriplegia with Traumatic Spondylitis.

By DOUGLAS McALPINE, M.B.

M. D., FEMALE, aged 42, was well until February, 1921, when she fell into an area basement. She immediately lost consciousness. Later she found she could not move her head or any of her limbs. She had retention of urine. At the end of one week power began to return in left arm, and gradually the other limbs recovered, but a general weakness has remained, this being most marked in her right arm. Since the accident she has suffered from shooting pains across the back of the neck and down both arms. She has shown progressive improvement whilst under observation.

Examination shows nothing abnormal in the cranial nerves. No localized wasting in shoulder or arm muscles. No sensory disturbance in limbs or trunk. Moderate loss of power in both arms, right more than the left. Tone in right arm is increased in triceps and flexors of the wrist. Posture of right arm when walking is one of extension at the elbow, pronation of the forearm, flexion at the wrist. No material change in tone or posture in left arm. Tone in the lower limbs is slightly increased in extensors, but power is quite good. Deep reflexes are much exaggerated in arms and legs, but more so on right side. Right plantar response is extensor, while left is equivocal.

X-ray examination shows bony outgrowth from third, fourth, fifth and sixth cervical vertebrae.

### Case of Syringomyelia showing Pain of Central Origin.

Shown by A. G. DUNCAN, M.B.

(For Dr. CAMPBELL THOMSON.)

W. M., MALE, aged 37. Fifteen months ago he first noticed a weakness in his left arm, which has gradually progressed. Pain was also experienced in left arm, and later in left upper chest and left side of the face, and this has persisted.

Examination shows hyperaesthesia to cotton-wool and pinprick, with partial loss to heat and cold in distribution of left fifth cranial nerve. In addition there is moderate loss to all forms of sensation on left side of neck, left arm and left trunk, as low as level of eighth dorsal segment. The loss of postural sensibility is very marked in left arm. Nystagmus present. No involvement of other cranial nerves. No muscular atrophy left arm. Deep reflexes more active on left side and left plantar response is extensor in type. There is no loss of sensation in left leg or on trunk or limbs on right side.

**Case of Right Fronto-parietal Tumour ; Cracked-pot Percussion Note over Right Frontal Bone ; Left Palmar Reflex.**

By GEORGE RIDDOCH, M.D., and W. RUSSELL BRAIN, B.M.

W. G., AGED 8, Hebrew boy, left-handed and a twin, was noticed six months ago to be weak in the left hand, which has become progressively weaker. He has complained of headaches beginning in the right temporal region for four months, and has vomited every few days for the same period. His face was noticed to be "on one side" two months ago; and there is a history of occasional stammering and some indefinite visual impairment.

On examination on February 20, 1923, there was a visible boss on the skull in the right temporal region divided by the right fronto-parietal suture. On percussion a cracked-pot sound could be elicited over the right frontal bone while the left yielded a "boxy" note. The fundi showed blurring of the edges of both discs with one diopter of swelling in the right. There was a left hemiparesis, the weakness affecting the facial movements most, the arm to a less extent, and the leg only slightly. Sensation was normal.

A fortnight later small clonic movements were observed in his left face and arm, and right leg.

Palmar reflex: It was noticeable that objects placed in the left hand were the more strongly clenched the more one attempted to pull them away. This reaction can still be obtained and is evoked especially by moving contact from the flexor surface of the fingers and especially of the first finger and thumb. The reaction is entirely involuntary, for the patient obviously makes strong efforts to relax his grasp. He is usually unable to extend his fingers but the reflex can be well evoked even when spasm of the fingers is largely eliminated by passive flexion of the wrist.

At the present time there is some loss of postural sensibility in the fingers of his left hand. Tactile discrimination, localization and sensibility are also defective in the left upper limb. There is now some secondary atrophy of both optic discs.

We are indebted to Dr. Campbell Thomson for permission to demonstrate this patient.

## Section of Neurology.

President—Dr. WILFRED HARRIS.

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### Hysterical Fits, with some Reference to their Treatment.

By LEWIS R. YEALLAND, M.D.

FROM a psychological aspect it may be argued that the genesis of the neuroses is identical, in whatever manner their manifestations may be expressed, and therefore to separate one particular group of symptoms from another with a view to directing attention to their treatment is erroneous. This argument is logical enough; but in practice the suggestive factor is primarily associated with most, if not all, forms of treatment whether the suggestion is directed to a supposed cause of the disorder or, as in the method about to be described, to the removal of symptoms. Attention is here centred on that group of symptoms of the neuroses which is characterized by paroxysms or seizures and commonly termed the fits of hysteria.

The modern conception of the genesis of the neuroses, unlike the ancient supposition that hysteria was the outcome of wandering of the womb, cannot be repudiated in the light of anatomical facts. An example of this is to be found in that method of psychotherapeutic treatment, the theory of which is, briefly stated, to ascertain the genesis of the neurotic symptoms (which is supposed to reside in repressed desires, of which the memory is too painful to be retained, and is therefore suppressed), to release the repressed desire, then to allow it to become attached to the idea of the physician, and finally to release the energy expressed in the repressed desire and transfer it to a socially useful aim. It is claimed for this method, however, that proper technique does not admit of any suggestive factor. If this is true, the high degree of technical skill essential to carry out the treatment must confine the performance of such a process to those only who possess unusual advantages. Again, the disastrous results, which are not uncommon, are attributed to defective technique; practical training is therefore rendered too dangerous to be undertaken. Moreover, the process of treatment occupies a long time, and much patience is requisite in seeing the same individual day after day for years. Further, the penalty, expressed in distasteful memories of symbolic interpretation involved in the reading of Freudian literature, will have the tendency of inhibiting some from a further study of a subject which so closely simulates a pathological sexual curiosity.

It may safely be stated that at present most patients suffering from paroxysms of the neuroses are treated during the paroxysm by the infliction of pain, such as supra-orbital pressure, strong faradism, &c.; but methods

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employed in the treatment of the seizures are of little value in the prevention of a recurrence. The benefits of physic, dispensed to combat the recurrence of hysterical fits, are, in all probability, to be found in the suggestive factor arising from the administration of drugs. It would be difficult to discover a less burdensome aid to suggestion. The results of such treatment are within the experience of most physicians and need not be further discussed.

#### CLINICAL OBSERVATIONS.

The method that I have adopted to prevent recurrences of the fits of hysteria is based on the following clinical observations:—

- (a) During a paroxysm the patient reacts to external stimuli.
- (b) A patient suffering from the disorder can be made to reproduce a seizure to order by a simple method.
- (c) The seizure thus produced corresponds in every apparent detail to the patient's previous attacks.
- (d) One type of seizure is constant in the individual, and there is no change from one type into another.
- (e) During a paroxysm which is reproduced to order the patient can be made to realize that consciousness is retained from the beginning to the end of the seizure.
- (f) The patient, after realizing that there is no loss of consciousness in the seizure, often becomes a good subject, in whom the power of restraint or inhibition can be re-educated.
- (g) After a short course of re-education frequently no further attacks occur.
- (h) There are definite types of seizure which render classification possible.

During a hysterical attack a patient reacts to external stimuli; for example, the convulsion will subside on the infliction of pain if the latter is severe enough. Again, the attacks become aggravated on the application of passive restraint; indeed it would appear that the severity of an attack varies conversely with the degree of force exerted in attempting to hold the patient down. Such reaction to external stimuli would suggest that the degree of loss of sensibility is not deep during a seizure. The depth of insensibility appears, from observation, to vary with the type of seizure rather than with the severity of the attack, the strongest stimuli being needed to rouse patients in whom tonic-like fits occur. Increase in the violence of the seizure on restraint would indicate that a seizure can be demonstrated to order; and this is further proved by the occurrence of an attack when the patient is sufficiently urged to demonstrate it.

In order to bring on a paroxysm in a patient subject to hysterical fits, considerable patience is sometimes needed, and patients vary remarkably in their willingness to produce an attack to order. Usually, at first, the patient will assert that it is impossible for him to demonstrate an attack as he does not know its nature; however, persistence in urging the patient will result in feeble attempts being made by him to describe the nature of the attack in words. In cases in which much difficulty is experienced in persuading the patient, methods may be employed to assist him to demonstrate the attack. For example, while standing he is commanded to close his eyes tightly and to make his body tense; while he lies on his back on a couch, he is ordered to raise his legs against the demonstrator's hands, which are placed on the patient's ankles. In the majority of cases an attack is readily reproduced by the latter method,

but in some cases constant persuasion, accompanied by determination, is needed. It is, of course, essential to secure the patient's confidence, otherwise he may think he is being ridiculed.

The character of the attacks thus produced resembles in every apparent detail the patient's previous attacks. This conclusion is arrived at by observing the seizure while the patient is waiting his turn for attention in the out-patient department of the hospital (where seizures are not uncommon, particularly on the first visit), and afterwards by persuading the patient to demonstrate a seizure to order. Again, friends of the patient who have observed previous seizures in the individual render further confirmatory evidence. Moreover, the patient will admit the similarity of the seizures after he has demonstrated an attack to order.

Although seizures vary in their character in different individuals, they always assume the same type in the particular individual, and there is no change from one type of seizure to another type. However, the severity of an attack in a given subject is rarely constant and often mild attacks alternate with strong ones.

During a paroxysm produced to order the patient is constantly spoken to and instructed to direct his attention to the general nature of the attack until it has subsided, after which he is asked to describe in words the various movements that were performed in the seizure. If his description appears to be hazy, the patient is again urged to reproduce an attack and this must be repeated until he has in general an intelligent conception of the nature of the movements. The patient is next asked to describe in detail by words the exact nature of the initial movements; if he cannot, an attack is repeatedly produced until he is able to do this. Finally, his attention is directed to the symptoms that preceded the initial movements, and he is asked to describe them. Some, on realizing that they are conscious during a seizure and that they have formed a clear idea of the nature of the attack from beginning to end, have no further seizures. But the majority of patients require a period of time during which re-education in their powers of inhibition must be undertaken. In order to accomplish this, my method is first of all to instruct the patient to lie on a couch and to urge him to have an attack; during that time he is instructed to raise the lower limbs against the resistance of my hands placed on his ankles. After the attack has subsided he is ordered again to go through the same movements, but this time he is urged to relax the body during his attempts to raise the limbs and to inhibit the tendency to make the body rigid. It will be observed that although the patient is attempting to carry out the movement, the limbs are rigid and raised only a few inches from the couch in spite of the fact that pressure exerted on the ankles is so slight as not to interfere with the performance of the movement. It is advisable to persist with the patient until he is able to raise the legs at right angles to the trunk. When this is accomplished the muscles of the limbs are no longer rigid and the patient becomes normally relaxed; the face no longer bears the expression of great strain, and there is reasonable ground for anticipating that the patient will successfully inhibit subsequent tendencies to seizures. The result is not always satisfactory and if the previous methods have failed it is necessary to attempt to break down the resistance of the patient by inducing attacks frequently. For example, if he is subject to three or four attacks a day, attacks to order must be brought on in excess of that number and diminished from day to day, and if necessary from week to week, this depending upon whether or not there is a return of the paroxysm in the interval. It will be

found that the patient will soon resent this treatment in view of its exhausting nature; this is an indication for still more strenuous efforts to conquer the patient by producing an even greater number of attacks and so to create in him an atmosphere of aversion to the attacks. The process, if successful, is usually not a long one and the resistance of the patient breaks down after the second or third attempt. This method of treatment should not be undertaken in Types 3, 6, and 7.

#### TYPES OF SEIZURES.

Seizures the outcome of the neuroses present many types, and it has been my experience, from clinical observation, to note the following principal types.—

- (1) An apparent momentary loss of consciousness—a faint.
- (2) A state of apparent coma lasting for hours, in which the pulse and respiration rates are normal.
- (3) A pseudo-tonic convulsion.
- (4) A pseudo-clonic convulsion.
- (5) A convulsion beginning in a pseudo-tonic contraction and followed by a pseudo-clonic contraction.
- (6) Hystero-epilepsy.
- (7) Attacks of uncontrollable laughing or crying in which the patient passes large quantities of urine.
- (8) Other violent movements expressed in attempts to injure himself or those who attempt to restrain the patient by holding him down.
- (9) Attacks of staggering.

Paroxysms of the neuroses are not uncommon in epileptics.

#### Illustrations.

*Type 1.*—An apparent momentary loss of consciousness is not uncommon in neurasthenic women whose symptoms are in the most part subjective. These patients complain of attacks of vertigo or fainting, in which they recognize their surroundings but are unable to speak. Other conditions which cause vertigo and fainting must of course be eliminated before a diagnosis of hysteria is made. From the history of some of these patients *petit mal* may be suspected and when an attack is not witnessed such a diagnosis is not infrequently made. The demonstration of an attack to order always confirms the diagnosis.

A woman aged 45, the wife of a hairdresser, who resented the imputation that her husband was a barber, complained of "neurasthenia." She did not volunteer information regarding the occurrence of attacks, and, when asked directly if she ever fainted, at first affirmed that she did not have hysteria and was annoyed. Soon, however, she admitted having attacks in which she "lost" herself for a moment and always in the presence of her husband. After the threat that she would leave the hospital and return to her home rather than submit to the indignity of demonstrating a seizure, her confidence was ultimately gained and she was at length persuaded to demonstrate an attack in which she became rigid, clutched her right forearm with her left hand, threw her head over the back of the chair on which she was sitting and began to respire noisily. Her face became slightly cyanosed and her eyes were closed. The attack lasted about fifteen seconds. Owing to failure on the part of an inexperienced nurse to inhibit a laugh, the patient rose quickly to her feet, left the room indignantly and has not since returned to the hospital. This case illustrates the fact that care



must be taken to gain and remain in the confidence of the patient, and shows the unfortunate results that ensue if hysterical manifestations are not treated with respect.

*Type 2.*—The *simulation of coma* as the outcome of the neuroses is, in my experience, not common in hospital practice. It may be argued that when patients assume a state of coma they should not be considered as hysterical, but it must be admitted that the patient at such a time is not normal mentally. The condition often occurs in young women who are unaccustomed to the ordinary knocks of life. They are usually irritable.

For example, a young woman, aged 22, was to be married in a week's time. One night just after dinner, the mother effected an entrance into the consciousness of the new relative in consequence of which the mind of the *fiancé* was impervious for a moment to a question asked by the patient, who, on the realization of the slight, retired from the room to be further submitted to the indignity of a lack of recognition of her absence for nearly an hour. She was found on her bed fully dressed and could not be roused. It was reported that she was pulseless but breathed slightly, and the diagnosis transmitted over the telephone was cerebral hæmorrhage. When I examined the patient in the presence of relatives she would not reply to any questions asked her, and no effort was made to rouse her by force. After the relatives had left the room she suddenly asked angrily, "Who asked you to come here?" The patient's confidence was soon obtained and she related the above-mentioned incident with much emotion. The patient has since become the mother of a healthy child and there has been no repetition of the attacks. In such cases, it is advisable not to acquaint the relatives with the nature of the disturbance.

*Type 3.*—A *tonic-like contraction* of the whole body, the history of which simulates the tonic stage of an epileptic fit, resembles clinically the first part of a hystero-epileptic seizure but does not progress further than a slight tilting forwards of the pelvis. The heels and occiput are pressed firmly against the couch, the body is rigid and the pelvis is raised from the surface of the couch. The face is flushed, the eyes closed tightly and there is an expression of severe distress. During this pseudo-tonic state the breath is held. After a half-minute to a minute the head turns to the side and the muscles of the body suddenly relax. The respirations then become very rapid and the patient either cries or laughs. The following account is an illustration of this:—

A chorus girl, aged 21, fell from some badly constructed steps on the stage at a rehearsal, injuring the back of her head, and had a seizure. There was no scar or other evidence of injury. She was unable the next day to take her part in the performance in view of another attack, which, when demonstrated to order, proved to be of the type described here. For two months the attacks occurred, on an average, twice a week and for a week previously to my seeing her an attack occurred every day. Considerable persuasion was needed in this case to produce an attack to order. Eventually an attack was induced, after the patient had been instructed to raise the limbs against passive resistance applied to the ankles. The patient's sister witnessed this attack, as she had other previous seizures, and confirmed its similarity in every detail to the other attacks. During the seizure the patient was spoken to and replied to questions asked her with considerable effort. When the attack had subsided she was asked whether she thought that further attacks could be prevented by her own effort, and she replied in the affirmative, stating that she had not previously recognized that she was conscious during the attacks. She returned to her work in the chorus in a week's time, and did not fail to attend either rehearsals or performances. When I saw the patient a month later she said that there had been no return of the attacks. Two months later a seizure occurred in bed, and she volunteered the information that it was difficult for her to restrain the seizure during the menstrual periods and that



she deliberately had an attack. She said that she could have inhibited this attack but chose a time for it when it did not interfere with her work. She thought that if she married it would not be necessary for her to have an attack, and asked advice regarding that matter. The patient has been married for nearly two years and there has been no recurrence of the seizures.

This particular case is chosen as an illustration because it might be regarded as favourable for psycho-analytic speculation, and it may be asked what treatment is to be employed in those cases where lack of opportunity renders marriage impossible? When the patient realizes that the condition resolves itself into one of conscious choice it would appear that there is no treatment except that which pertains to the practice of restraint on the part of the patient. These patients return to their duties and, although attacks sometimes occur in secret, their productive capacity is not interfered with and they become useful citizens.

*Type 4.*—*Clonic-hysterical fits* are more common in men and in my experience form the only type of hysterical seizure that occurred in soldiers in the recent war. A localized tremor is usually present either in the fingers or the tongue, or there may be a blepharospasm. The attack, as a rule, comes on with some excitement or sudden shock and often the banging of a door is sufficient to bring on a seizure. The tremor, which at first may be fine, becomes coarse and it soon spreads all over the body and the patient falls down in a coarse generalized tremor and appears to be unconscious. As a rule, he does not become violent unless he is passively restrained, in which case the convulsions become greatly aggravated.

A sawyer, aged 24, was unable to work for five years on account of seizures, which began during an air raid in 1916, and one or more attacks occurred almost every day. There was a history in this case of mutism which persisted for six months after the first seizure, to be followed by stammering. A partial right hemianesthesia was made out. The patient responded to the suggestion to demonstrate an attack without much persuasion. This attack, when fully matured, consisted of violent clonic movements of voluntary muscles including the face, neck, trunk and limbs. During the first attack demonstrated to order the patient did not respond to questions asked him, and after it had subsided he was unable to describe its nature. During the second attack produced to order the patient replied to questions during the first part of the attack but when the movements became violent he did not answer questions put to him. When he was seen a week later he had had no attacks in the interval, but complained of severe fatigue resulting from the attacks produced during his former visit. He protested against further attacks being produced, but he submitted eventually, and in this attack, he was able to respond to questions asked him throughout and afterwards was able to describe in detail the nature of the attacks. The following week, during an attack produced to order, he was instructed to relax himself as soon as the tremor became generalized, by allowing his arms to fall loosely at his sides. A year after this treatment, when the patient was heard from, there had been no recurrence of the seizures and he had been employed for nearly that length of time in a remunerative position. Sometimes, he said, he had a feeling of an attack impending, but by relaxing himself he was always able to prevent it. It should be stated that this patient was of fairly average intelligence, in which case a good result is to be expected.

*Type 5.*—*Seizures beginning in tonic contractions and followed by clonic contractions* are not uncommon in men, and this type is most frequently mistaken for epilepsy. The patient suddenly becomes rigid when standing, his trunk is erect and his head thrown back; he staggers backwards or lateral-wards, and finally falls, shaking all over. He may or may not injure himself when he

falls. This type resembles in many details the one just described, but differs from it in an absence of the initial tremor and in the presence of a definite initial rigid state.

A grocer's assistant, aged 55, lost his wife, who died suddenly from heart failure. On hearing the news he collapsed, and was thereafter subject to seizures, which, according to his local doctor's description, appeared to be correctly diagnosed as epilepsy. The attacks occurred, on an average, once a day. When the patient was asked to describe in words the nature of the attack, suddenly his eyeballs rolled upwards, his head was thrown back, and he remained rigid for about ten seconds. Then he began to stagger backwards and to shake all over, and fell bruising his right forearm. A nurse in attendance tried to prevent him from falling, and on my instructions attempted to hold him down after he had fallen. With this restraint the violence of the shaking became aggravated. The attack began to subside with the removal of the restraint. With renewed passive restraint the movements again became violent, so that with repeated application of restraint the condition simulated *status epilepticus*, but it differed from the latter in the absence of a repetition of the rigid stage. When the restraint was removed altogether and the patient left to himself the paroxysm ceased, and when he was instructed to rise to his feet he did so with difficulty, and staggered. When seen next time the patient was urged to produce another seizure, and with some persuasion he succeeded in demonstrating an attack which was in all respects similar to the preceding one. Again he fell and injured his right forearm. After the attack had subsided he was urged to demonstrate another seizure, and resented the suggestion because of the pain in his right forearm. However, he submitted, but this time when he fell he did not injure himself. On this occasion four attacks were induced, and afterwards he was instructed to return in a week's time and was informed that more attacks would be brought on if in the interval there was a recurrence. When he returned a week later, in spite of the absence of attacks in the interval, two attacks were induced, much to the patient's distress. The following week one attack was brought on, although there had not been a repetition of the seizures. The result was completely satisfactory. He was treated over a period of six weeks, and there has been no recurrence of attacks.

*Type 6.*—*Hystero-epilepsy* or *grande hystérie* as described in detail by Charcot and Richer, is not common in this country, and my experience of this type of hysteria is confined to four cases, only one of which was treated by the methods described here. The patients were all women. In view of the spectacular nature of the attack and the common idea that the state is partly epileptic, a record of observations made in one of the cases will be given. The attack was induced in the patient and witnessed by three colleagues.

The case was that of an unmarried woman, aged 26, who first associated herself with these attacks at the age of 18. The attacks occurred, on an average, twice a week. The patient claimed to be entirely ignorant of their nature, and she was therefore unable to give a description of the attacks. On one occasion she was incontinent during an attack when it occurred in bed, and her tongue was sore after the attack. The patient was sent into a dressing room with the order to place herself on a couch there and to demonstrate the attack. The order was at first resented, but she was eventually persuaded after complaining that the couch was too narrow for the performance! The attack began by the patient throwing herself into a marked degree of generalized tension, as if she were attempting to increase her stature by an apparent stretching of her neck. There was forcible plantar flexion of the feet, so that their plantar surfaces approximated to the surface of the couch, the legs being drawn tightly together. The arms were drawn close to the sides of the body, and they also appeared to be lengthened by over-extension of the wrists and fingers, the forearms being at the

same time distinctly pronated so that the dorsal surfaces of the thumbs approximated to the surface of the couch. The eyes turned upward, the sclerae only being exposed: the face almost from the beginning became distorted in grimaces, and the cheeks and lips were firmly pressed against the gums and teeth. The patient's explanation of the movements of the face at this stage was to the effect that such contortions assisted in the production of saliva. Slowly the pelvis was raised from the surface of the couch with the assistance of her thumbs, and at the same time the head was gradually thrown backward. The soles of the feet now touched the surface of the couch and were firmly pressed against it. The arms began to draw away from the sides of the body. Slowly the head retracted until the forehead rested on the surface of the couch. This was accomplished by the patient tilting the pelvis more and more ventral-wards by drawing the feet under the trunk towards the head, until finally the forehead and the heels became approximated. The face was by this time intensely cyanosed, and the front of the neck appeared distinctly swollen, the cyanosis being due to partial suffocation owing to the position of the head. Respirations became quickened, and the patient could be seen to protrude blood-stained foam between her teeth by forcibly pressing the tongue against them. The arms assumed a position at right angles with the trunk, and athetoid movements appeared in the fingers. The explanation given by the patient of the position of the arms was to the effect that it helped her to balance herself. When the arms were forcibly moved in all directions they appeared to be wax-like, and the patient's equilibrium was not upset by these movements. The arched body was extremely tense and supported without difficulty pressure on the abdomen. In this state of *arc de cercle* the patient answered questions asked her by others in the room, but to the question, "You do not know what I am saying?" asked by one of my colleagues, who at this stage came in, she did not reply. The patient remained in this position for ten minutes, clonic movements occurred for a few seconds as the body tilted over to the right side, and gradual relaxation from superextension slowly took place, until the patient once more assumed a relaxed supine position. The plantar surfaces of the feet on the couch, together with the forehead, formed a tripod during the severe state of opisthotonus, and seemed to be sufficient to support the body in that position. With complete relaxation there was extreme fatigue. In the course of this attack there was no appearance of the third and fourth stages, as described by Charcot, in which dramas are enacted, and a period of delirium occurs. The treatment of the seizure consisted in demonstrating to the patient that she was not, as she supposed, unconscious during the attack, but, rather, fully alive to every detail. When the patient returned in a fortnight she reported the occurrence of one paroxysm when she was in bed. On being told that the attack could be controlled the same as walking could be controlled, she admitted that the attack had been deliberately induced by her because it produced a "pleasant sensation." By degrees she chose to inhibit the impulse to produce the sensation, but not altogether. The attacks have been reduced in the last two years from several a week to about the same number a year, and the paroxysms occur now when she is alone and do not interfere with her duties.

*Type 7.—Paroxysms of uncontrollable laughing or crying in which large quantities of urine are passed, in my experience only occur in women who practise uncleanness with themselves. Attempts to induce an attack in these cases are of course to be avoided. Such patients are benefited by hard manual labour on farms.*

*Type 8.—Seizures, which may be described as violent movements in which the patients tear at their clothing, attempt to injure those who make an effort to restrain them and, in the event of nobody attempting to do so, pull at their own hair and injure themselves by scratching or biting their arms, are not uncommon in both sexes but are much more frequent in females. The movements are very grotesque and the patients in their agitation will expose themselves by shouting out or calling for a friend. The condition is sometimes difficult to diagnose at first sight from manic-depressive insanity. The seizure in no way resembles epilepsy.*

A woman, aged 30, suffered from this type of attack for a year and seizures occurred at an average of three times a week. The first paroxysm began after she was told that she had contracted syphilis from her husband and while she was waiting for an intravenous injection of novarsenobillon. The attack was very violent and she was held down by fellow-patients in the out-patient waiting room. A week later, when she was urged to demonstrate an attack, she began it by turning her trunk and head violently from left to right. She threw out her arms and called for her mother and knocked her head against the floor. Her hair soon became dishevelled and she began to tear at her clothing shouting louder and louder. She was firmly ordered to rise to her feet, which she did and complained that her arm was numbed. She made a promise to control these attacks and there has been no recurrence for six months.

*Type 9.—Attacks of staggering*, which according to the subjective evidence gained from the patient would suggest *petit mal*, are often preceded by *globus*, palpitations or disagreeable sensations in the abdomen. The attack may occur in the absence of these symptoms or one or all of them may be present.

Thus, a joiner, aged 48, "came over giddy" when working on a building in a safe part and "lost his senses" but did not fall. There were no signs of organic disease and other causes of vertigo were eliminated before a diagnosis was arrived at. When he was asked to demonstrate an attack he said he became giddy and lost himself completely but could not demonstrate the nature of the attack. After much persuasion and after he had paced up and down the room in his attempt to produce an attack for several minutes, he suddenly began to stagger sideways and to the right, reaching out his right hand for the purpose of supporting himself at a table close by. The patient said he was giddy and that the attack which had just occurred was not the outcome of efforts to demonstrate the attack. He was at once ordered to produce another seizure, a suggestion to which he almost immediately responded. The attacks were repeated several times and he was finally convinced that he was completely conscious during the attacks and that it was in his power to bring on an attack at any time, as it was under the control of his will. This resulted in a subsidence of the attacks for eight months; since that time there has no note been made of his case.

Epileptics may be the subjects of paroxysms of the neuroses. A hysterical attack may immediately follow an epileptic seizure, or the former may bear no relation to the latter as far as its occurrence in time is concerned. Again, the occurrence of *petit mal*, *grand mal* and paroxysms of the neuroses are not uncommon in the same individual. The conditions must be treated separately. These patients should be given luminal,  $1\frac{1}{2}$  gr., three times a day, in addition to the treatment for the hysterical condition.

#### CONCLUSION.

A diagnosis between hysterical fits and epilepsy is difficult and sometimes impossible unless a seizure is witnessed. Babinski and Froment were able, in the case of one patient, to reproduce a hysterical fit by suggestion aided by electricity. Electricity is certainly not needed to reproduce a hysterical fit and it is doubtful whether extraneous suggestion is needed. I have frequently seen hysterical fits reproduced when the patient has attempted to raise the lower limbs against passive resistance, no order having been given and no comment made, other than "raise the lower limbs."

I have the permission of an officer who was on duty at the recent Trooping of the Colours to state that 5 per cent. of the rank and file, after standing in a rigid state of attention, fell out in a fit in the course of the first ten minutes of the performance. An attack always seems to follow a certain state of tone of the muscles, which is created by simultaneous contraction of agonists and

antagonists. Clonic movements occur in many neurasthenic individuals subjected to the same test, and in them there is the same distressed facial expression. The hysteric makes no wholesome effort to overcome the passive resistance, whereas the healthy individual usually either succeeds or, in his attempt to do so, sits upright. This test is, I believe, of considerable diagnostic value and may be as easy or as difficult to elicit as a plantar reflex.

The treatment which was considered in these cases resolves itself into the removal of symptoms. In the selection of 100 cases for the purpose of determining results, an equal number of each type of paroxysm has been chosen as far as that has been possible. The record has been brought up to a recent date and the period of freedom from the attacks has been found to vary from five years to six months. The percentage of recoveries from attacks is about sixty. Types 3, 6 and 7 materially diminished the number of apparent "cures," but in them improvement was observed in the marked reduction in the number of seizures and in the fact that the attacks occurred when the patients were alone.

